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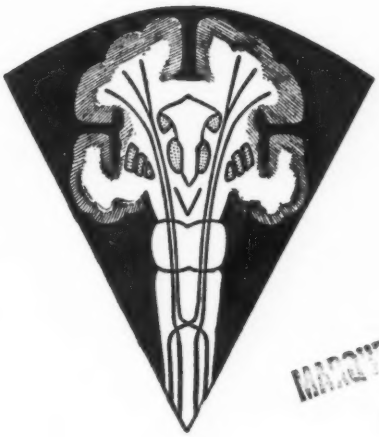

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[*J. Neurosurg.*]

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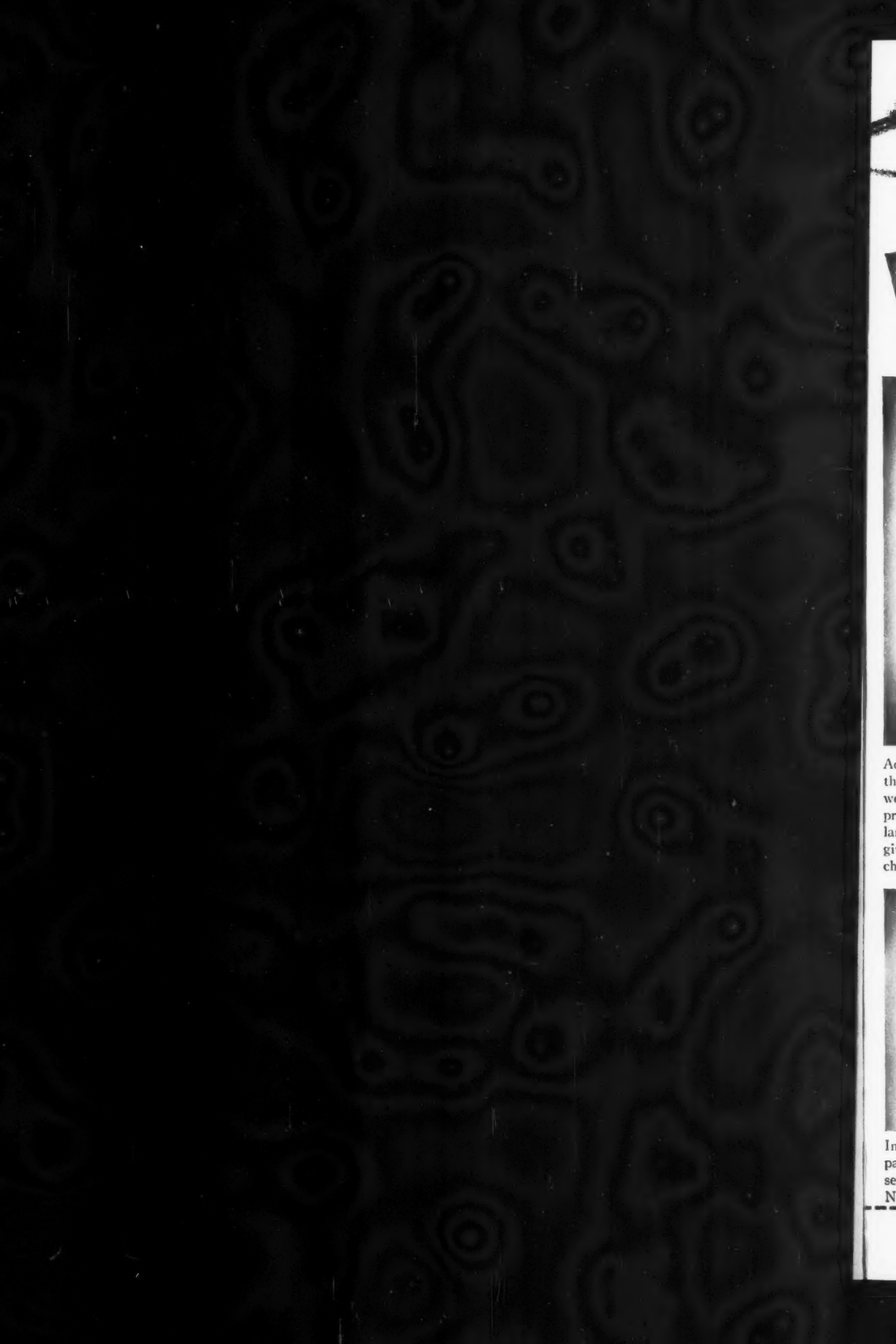

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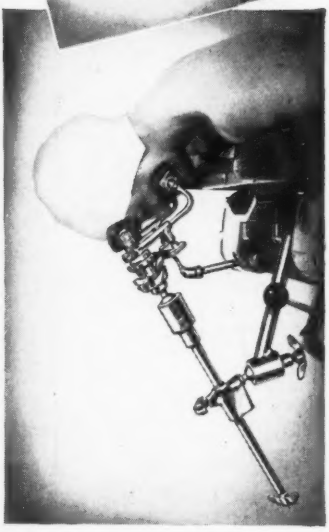

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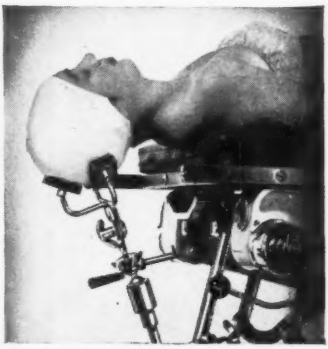

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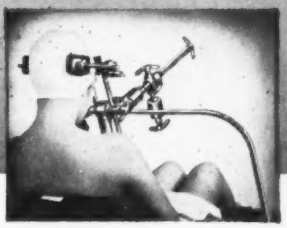

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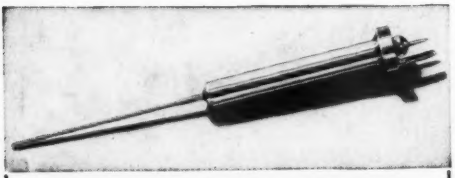

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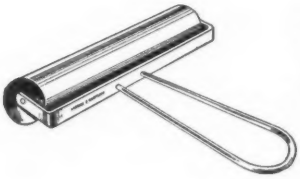

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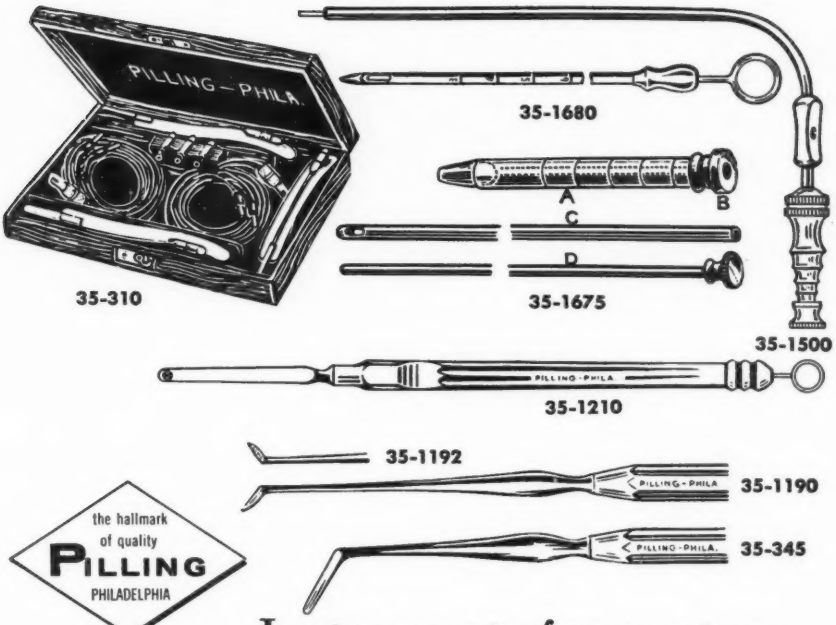

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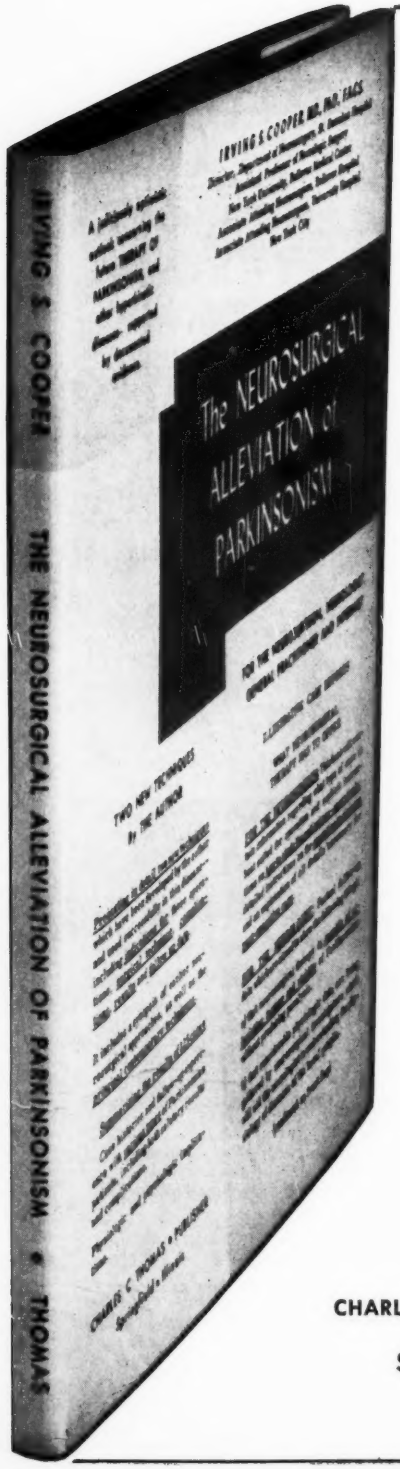

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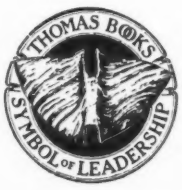

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J. C. Bacala, M.D.

(Abstracted from the Western Journal of Surgery, Obstetrics, and Gynecology, 64:88 (1956)

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NOTICE

Because of an unfortunate printer's error, the July 1956 issue of *Journal of Neurosurgery* (Vol. XIII, No. 4) contains wrong page numbers. The over-all pagination should be 311 through 412, instead of 223 through 324. The correct page numbers are:

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As a safeguard against future reference and for indexing purposes, we recommend that you correct the page numbers in the July 1956 issue by crossing out the present folios and inserting the new page numbers *in ink*.

We regret the error and any inconvenience the wrong page numbers might cause you.

ASTROCYTOMA OF THE BRAIN AND SPINAL CORD

A REVIEW OF 176 CASES, 1940–1949*

LAURENCE F. LEVY, M.D., AND ARTHUR R. ELVIDGE, M.D.
Department of Neurology and Neurosurgery, McGill University and the Montreal Neurological Institute, Montreal, Canada

(Received for publication February 14, 1956)

THE present survey is based upon 176 cases of astrocytoma treated in the 10-year period of 1940–1949 inclusive. They will be considered in relation to the anatomical location of the tumours: cerebral hemisphere proper (113 cases), 3rd ventricle (5 cases), optic nerve (3 cases), cerebellum (44 cases), and spinal cord (11 cases). The tumours of the optic nerve have been separated because of their peculiar location, while those of the 3rd ventricle are considered separately because of the special problems associated with their operative removal. Of the 165 intracranial astrocytomas, 73 per cent were supratentorial and 27 per cent were in the posterior fossa. Other authors show similar figures.

These astrocytomas have been subdivided according to the classification of Elvidge, Penfield and Cone[2] into piloid, gemistocytic and diffuse types.

The 113 tumours of the cerebral hemisphere consist of 58 (51 per cent) piloid, 27 (24 per cent) gemistocytic, 23 (20 per cent) diffuse and 5 (5 per cent) borderline astrocytomas. This latter group consists, histologically, of a tumour the largest part of which is an astrocytoma, but which, because of certain areas that are not typical, could not be classified properly in any of the three astrocytoma subgroups mentioned above. Nevertheless, they have been included in the series and therefore are classified in this additional subgroup.

The 5 tumours of the 3rd ventricle were all piloid astrocytomas, as were the 3 optic nerve tumours and all the tumours of the cerebellum, brain stem and spinal cord.

Follow-up reports are complete on 93.4 per cent of the patients. The longest follow-up period is 14 years and the shortest is 4 years. Excluding those lost to follow-up, 36.8 per cent of the patients are still alive and 63.2 per cent are dead.

I. SUPRATENTORIAL TUMOURS

1. PILOID ASTROCYTOMAS

There were 58 piloid astrocytomas of the cerebral hemispheres (not including the 3rd ventricle). Thirty-two of the patients were males, and 26 were

* A series of 106 cases in the 10-year period of 1928–1939 has been reviewed by Elvidge and Martinez-Coll.[1]

females. The average age at the time of admission was 33 years, 7 months (Table 1), and 29 years, 6 months at onset of the first symptom.

There was a history of convulsions in 43 (74 per cent) cases. Headache was complained of by 32 (55 per cent) patients. Papilloedema occurred in 25 (43 per cent) patients.

The average pre-operative duration of symptoms in all patients was 49

TABLE 1

Age incidence—piloid astrocytoma of cerebrum (58 cases)

No. of Cases	5	5	15	11	15	5	2
Age (years)	1–10	11–20	21–30	31–40	41–50	51–60	61–70

months. The longest was a 30-year history of convulsions, while the shortest was of convulsions of 2 months' duration.

The localization of the tumours is given in Table 2 and the relationship to convulsive seizures is shown in Table 3.

Fifty-eight surgical specimens were re-examined. Twenty of these contained macroscopic cysts (34 per cent) while a further 7 had microscopic cysts (12 per cent). The incidence of convulsions is not related to the presence of cysts. Of the 20 patients with macroscopic cysts, 14 (70 per cent) had convulsions. The incidence of seizures in those patients with cysts is therefore not different from the incidence of seizures in the entire group (74 per cent).

Calcium was present in 8 (14 per cent) histological specimens. Although calcium was seen by roentgen ray in 9 cases, in only 6 of these was there microscopic evidence of calcium in the tumour. In the other 2 cases in which calcium was found in the tumour there was no sign of it in the roentgenograms. Thus the total number of patients with calcification was 11 (19 per cent).

Four tumours showed some sign of increased malignancy (endothelial hypertrophy, pleomorphism, or giant cells, etc.), and mitoses were found in 12 (19 per cent). Satellitosis was present in 3 tumours.

Operative Summary. Biopsy only was performed on 5 patients and was preceded by Torkildsen's procedure in 1 of these. Further resection was withheld in these patients for the following reasons:

No. 6. Tumour deep in motor and speech areas.

No. 18. Tumour deep in basal ganglia. Patient died on day of biopsy.

No. 21. Tumour deep in basal ganglia of a boy aged 14 with ventricular obstruction which had been relieved by Torkildsen's procedure.

No. 101. Frozen section of biopsy in an elderly patient showed a malignant glioma.

No. 104. After biopsy patient became aphasic and hemiparetic, and remained so for another year.

There was 1 operative death in this group of 5 cases.

The remaining 53 patients underwent surgical removal of all or part of the tumour. In 5 cases (Nos. 50, 63, 64, 68 and 100), the tumour was seen to

TABLE 2

Location—piloid astrocytoma of cerebrum (58 cases)

Location	No. of Cases	Per Cent
Frontal	23	40.0%
Temporal	7	12.0%
Parietal	5	8.6%
Frontoparietal	4	6.0%
Temporoparietal	3	5.0%
Basal ganglia	3	5.0%
Frontotemporal	2	3.4%
Parieto-occipital	2	3.4%
Corpus callosum and bilateral	2	3.4%
Post-temporo-occipital	2	3.4%
Frontoparieto-occipital	1	1.7%
Frontohypothalamus	1	1.7%
Interventricular	1	1.7%
Cingulate gyrus	1	1.7%
Frontoparietotemporal	1	1.7%
Right-sided	24	
Left-sided	31	
Bilateral	3	

have crossed the midline to the other side. It might be thought that crossing of the tumour to the other side would be of poor prognostic significance. This, however, is not necessarily so. Three of the patients lived 5 years (No. 63), 3 years (No. 64), and 8 months (No. 68), respectively, after operation, but 2 died in the immediate postoperative period. Eight of the 53 patients were reoperated upon in the course of their immediate postoperative period.

Nine of these 53 patients died in the immediate postoperative period (including 3 of the 8 who were re-explored). Five patients had a further excision of tumour 2 to 4 years later. There were no postoperative deaths in this latter group.

Thus, a total of 73 operative procedures were performed on 58 patients with 10 operative deaths, a case mortality of 17 per cent.

TABLE 3

Relation of seizures to region affected—piloid astrocytoma of cerebrum (58 cases)

Region	No. of Cases	Major Seizures	Minor Seizures	Total Seizures	Percentage
Frontal	23	18	2	20	87%
Temporal	7	5	1	6	85%
Posterior temporal, parietal and occipital	12	6	—	6	50%
Deep and midline	8	3	2	5	62%
Frontoparieto-occipital	5	4	—	4	80%
Frontotemporal, frontoparieto-temporal	3	2	—	2	66%
	58	38	5	43	

The fact that a removal is known to be incomplete does not of necessity indicate a poor prognosis, but only that it was difficult for the surgeon to determine the limits of the tumour at the time of operation.

Of 38 survivors after operation (6 of the 44 survivors were lost to follow-up), 16 had what was thought to be a complete removal, and 22 an incomplete removal. The average survival of the 16 patients who had a so-called complete removal (ignoring the question of roentgen-ray therapy and including 6 patients who are still alive), is 3 years and 10 months. The average survival of the 22 patients who had an incomplete removal, including 10 who are still alive, is 4 years and 2 months. The survival and morbidity data are shown in Tables 4–10 inclusive.

In summary it may be said that of the original 58 patients, 10 (17 per

TABLE 4

Survival after biopsy and surgical removal—piloid astrocytoma of cerebrum

Biopsy Only—4 cases

Case No.	Survival	Case No.	Survival
6*	3 yrs. 9 mos.	21	9 mos.
104*	1 yr. 2 mos.	101	1 mo.

Surgical Removal (dead)—21 cases

Case No.	Survival	Case No.	Survival	Case No.	Survival
41*	7 yrs.	27	3 yrs. 7 mos.	70	1 yr.
22	5 yrs. 6 mos.	58*	3 yrs. 4 mos.	108	1 yr.
5*	5 yrs.	64	3 yrs.	59	1 yr.
80*	4 yrs. 8 mos.	52*	2 yrs. 8 mos.	43	11 mos.
45	4 yrs. 3 mos.	13	2 yrs. 7 mos.	68	8 mos.
63	4 yrs. 3 mos.	20*	1 yr. 9 mos.	73	6 mos.
112	4 yrs.	46	1 yr. 6 mos.	99	5 mos.

Surgical Removal (alive)—17 cases

Case No.	Survival	A.S.E.†	Case No.	Survival	A.S.E.†
47	8 yrs.	333	84*	5 yrs. 3 mos.	223
56*	7 yrs. 6 mos.	334	82	5 yrs. 2 mos.	434
76*	7 yrs.	333	109*	4 yrs. 9 mos.	334
69*	6 yrs. 6 mos.	434	95*	4 yrs. 8 mos.	443
88*	5 yrs. 11 mos.	444	105	4 yrs. 4 mos.	444
87*	5 yrs. 7 mos.	433	29	4 yrs. 4 mos.	220
86	5 yrs. 6 mos.	330	110*	4 yrs.	220
79*	5 yrs. 5 mos.	334	111	4 yrs.	444
97	5 yrs. 3 mos.	223			

* Received roentgen-ray therapy.

† The A.S.E. classification after Penfield (A=anatomical, S=symptomatic, E=economical): 4 is a perfect score; 0 is complete failure. A perfect score means there is no major organic symptom or sign and the patient can work.

cent) died in the immediate postoperative period, and 6 were lost to follow-up. Of the 42 who were followed, 4 had biopsy only, and 38 had removal of tumour. Of these latter, 4 are normal and self-supporting; 10 are disabled but self-supporting; 3 are disabled and not self-supporting; and 21 have succumbed but of these 7 were self-supporting before death.

2. ASTROCYTOMA DIFFUSUM

There were 23 cases in this group. Twelve of the patients were males, and 11 were females. The average age was 35 years at the time of admission (Table 11) and 31 years 7 months at the commencement of symptoms.

The average pre-operative duration of symptoms was 41 months.

Nineteen (82 per cent) patients were admitted with a first complaint of seizures. Ten (43 per cent) of these had no other complaint beyond their seizures and of these 10, 3 had no abnormal neurological findings. Nine (39 per cent) patients suffered from headache, and papilloedema was found in 6

TABLE 5

Comparative survivals—piloid astrocytoma of cerebrum

1. Average survival of all patients (except those with biopsy only) regardless of treatment (17 living, 21 dead)—4 years.
2. Average survival after surgery alone (7 living, 15 dead)—3 years 2 months.
3. Average survival after surgery and roentgen-ray therapy (10 living, 6 dead)—5 years.
4. Average survival of 18 patients with macroscopic cysts (12 living, 6 dead—10 received roentgen-ray therapy, 8 did not)—4 years 6 months.
5. Average survival of 8 patients with macroscopic or microscopic calcium (5 alive, 3 dead—1 other lost to follow-up, 2 died at operation)—4 years 6 months.
6. Average survival of 7 patients with tumours showing mitotic figures (4 dead, 3 living)—4 years 9 months.
7. Average survival of 4 patients with tumours noted as being more than usually malignant (2 dead, 3 living)—3 years 8 months.
8. Five-year survival: 14 (3 dead, 11 living) of 38 patients (not including those with biopsy only)—37%. (Two more patients are alive 4 years or more after operation and are normal—if they are included, the 5-year survival rate rises to 42%).

TABLE 6

Average survival relative to site of tumour—piloid astrocytoma of cerebrum (42 cases including 4 of biopsy only)

Region	No. of Patients	No. Living	No. Dead	Average Survival (All Cases)
Temporal	6	4 (66%)	2 (33%)	4 yrs. 10 mos.
Frontoparietal	4	2 (50%)	2 (50%)	4 yrs. 1 mo.
Temporoparietal, temporo-occipital, parieto-occipital, parietal	7	1 (14%)	6 (86%)	3 yrs. 10 mos.
Frontotemporal, frontotemporoparietal	3	0	3 (100%)	3 yrs.
Frontal	17	6 (35%)	11 (65%)	3 yrs. 7 mos.
Deep and midline	5	0	5 (100%)	2 yrs. 10 mos.

TABLE 7

Survival by age group—piloid astrocytoma of cerebrum (38 cases)

Age Group (Years)	No. Alive	Average Survival to Date	No. Dead	Average Survival	Total Cases	Average Survival to Date
4–10	4	6 yrs.	1	3 yrs.	5	5 yrs. 5 mos.
11–20	3	4 yrs. 5 mos.	1	6 mos.	4	3 yrs. 5 mos.
21–30	2	5 yrs. 8 mos.	7	2 yrs. 1 mo.	9	2 yrs. 11 mos.
31–40	4	6 yrs. 5 mos.	5	3 yrs. 10 mos.	9	4 yrs. 11 mos.
41–50	4	4 yrs. 7 mos.	5	2 yrs. 5 mos.	9	3 yrs. 5 mos.
51–60	0		2	4 yrs. 3 mos.	2	4 yrs. 3 mos.
	17		21		38	

TABLE 8

Mortality after surgery—piloid astrocytoma of cerebrum (21 cases)

No. of Deaths	7	2	3	3	4	1	1
Per Cent	33%	9%	14%	14%	19%	5%	5%
No. of Years after Surgery	1	2	3	4	5	6	7

TABLE 9

Morbidity in 17 cases of cerebral piloid astrocytoma—patients now living (over 4 years)

Case No.	Present Condition of Patient	A.S.E. Code*	Survival
Normal—4 (24%) cases			
56	Occasional headaches	444	7 yrs. 6 mos.
88	Has baby of 4 mos. Gets occasional headache; has peri-carotid calcification following arteriography	444	5 yrs. 11 mos.
105	Labile personality; works at home and outside	444	4 yrs. 4 mos.
111	Arm gets tired easily; otherwise asymptomatic	444	4 yrs.
Residual Disability but Self-Supporting—10 (59%) cases			
82	Slightly dysarthric	434	5 yrs. 2 mos.
79	No seizures since second operation; mentally retarded	334	5 yrs. 5 mos.
109	Hemaniopic; goes to college	334	4 yrs. 9 mos.
95	Personality difficulties and loses time at work therefrom	443	4 yrs. 8 mos.
87	Has some seizures	433	5 yrs. 7 mos.
76	Goes to school, slightly backward	333	7 yrs.
47	Residual minor hemiparesis	333	8 yrs.
86	Occasional seizures; hemianopic	333	5 yrs. 6 mos.
97	Hemiparesis; looks after her home	233	5 yrs. 3 mos.
84	Occasional seizures; hemiparetic; poor convergence	223	5 yrs. 3 mos.
Residual Disability and not Self-supporting—3 (17%) cases			
29	Has convulsions; awaiting admission to School for Blind	221	4 yrs. 4 mos.
69	Has daily seizures; hemiparetic; does not attend school	220	6 yrs. 3 mos.
110	Hemiplegic; needs constant nursing	220	4 yrs.

* A=anatomical, S=symptomatic, E=economical: 4 is a perfect score; 0 is complete failure. A perfect score means there is no major organic symptom or sign and the patient can work.

TABLE 10

Morbidity in 21 cases of cerebral piloid astrocytoma—patients now dead

No. of Patients	Survival	Postoperative Condition
7	Over 2 yrs.	Were self-supporting and leading relatively full lives
6	Over 2 yrs.	Unknown
8	Less than 2 yrs.	Unknown

TABLE 11

Age incidence—astrocytoma diffusum (23 cases)

No. of Cases	1	0	4	14	3	1
Age (years)	1–10	11–20	21–30	31–40	41–50	51–60

(26 per cent) patients. Pyramidal signs or paresis of some degree were among the most common findings. Signs may be quite minimal. One patient (Case 96) of 50 years who had had convulsions for 32 years had only loss of weight and poor convergence at the time of first examination, although his symptoms were those of increased intracranial pressure.

The general location of the tumours is shown in Table 12 and the relationship of seizures to the region affected in Table 13.

Twenty-three surgical specimens were re-examined histologically. A microscopic cyst was present in 1, calcium was seen in 3 and mitoses were observed in 4.

TABLE 12

Location—astrocytoma diffusum (23 cases)

Location	No. of Cases	Per Cent
Temporal region	10	43%
Frontal region	8	35%
Frontoparietal	5	22% (1 had crossed the midline)
Right-sided	14	
Left-sided	9	

TABLE 13

Relation of seizures to region affected—astrocytoma diffusum

Region	No. of Cases	Major Seizures	Minor Seizures	Total Seizures	Percentage
Temporal	9	8	1	9	100%
Frontal	8	4	1	5	62%
Frontoparietal	5	4	0	4	80%

Operative Summary. Biopsy only was performed on 2 patients. One (No. 49) received deep roentgen-ray therapy and is still working in the post office, 7 years and 10 months later. The second patient (No. 23) survived 9 years.

Surgical removals were undertaken on 21 patients. One patient died in the postoperative period.

Two patients had further operations. One was for a recurrence with a spread across the midline. A further removal was carried out and the patient (No. 37) is now alive and well without any sign of recurrence $8\frac{1}{2}$ years later. In the second case a further exploration was made but no removal of tumour was carried out.

A total of 27 operations was performed on 23 patients with 1 death. Case mortality was 4.34 per cent. In 5 cases in which the removal was believed to have been complete the average postoperative survival was 1 year and 10 months (5 dead, 0 living). Two of these patients had roentgen-ray therapy after surgery, while 1 had had a negative exploration followed by a very small roentgen-ray dose elsewhere, prior to surgery here. In 15 cases a known incomplete removal was performed and the average length of life after operation was 3 years and 7 months. Ten of these patients received postoperative roentgen-ray therapy (12 dead, 3 living).

Follow-up studies were complete on 22 patients. The survival and morbidity figures are shown in Tables 14–20 inclusive.

TABLE 14

Survival after biopsy and surgical removal—astrocytoma diffusum

Dead—17 patients

Case No.	Survival	Case No.	Survival
23	9 yrs. (biopsy only)	8	2 yrs. 2 mos.
39*	8 yrs. 4 mos.	40	2 yrs.
10*	7 yrs. 9 mos.	96*	1 yr. 4 mos.
83*	3 yrs. 9 mos.	98*	1 yr. 2 mos.
36*	3 yrs. 1 mo.	17	1 yr. 2 mos.
92*	3 yrs.	31*	10 mos.
103	2 yrs. 10 mos.	54*	8 mos.
51	2 yrs. 8 mos.	53	5 mos.
		42*	3 mos.

Alive—5 patients

Case No.	Survival	A.S.E.†	Case No.	Survival	A.S.E.†
37*	9 yrs. 9 mos.	434	90*	5 yrs. 3 mos.	334
49*	7 yrs. 10 mos. (biopsy only)	434	106*	4 yrs. 6 mos.	434
			107	4 yrs. 3 mos.	220

* Received roentgen-ray therapy.

† A=anatomical, S=symptomatic, E=economical: 4 is a perfect score; 0 is complete failure. A perfect score means there is no major organic symptom or sign and the patient can work.

TABLE 15

Comparative survivals—astrocytoma diffusum (22 cases)

1. Average survival regardless of treatment (17 dead, 5 living)—3 years 9 months.
2. Average survival after surgery alone, including 1 biopsy (7 dead, 1 living)—3 years.
3. Average survival after surgery and roentgen-ray therapy (9 dead, 4 living)—4 years 3 months (excluding case No. 98).
4. One patient with a macroscopic cyst survived 8 years 4 months.
5. Average survival of patients with macroscopic calcium (3 dead)—1 year 6 months.
6. Average survival of patients with tumours showing mitotic figures (2 living, 2 dead)—5 years 11 months.
7. Five-year survival: 6 patients (3 dead, 3 living)—27%.

TABLE 16

Average survival relative to site of tumour—astrocytoma diffusum

Region	No. of Patients	No. Living	No. Dead	Average Survival (All Cases)
Frontal	8	0	8 (100%)	3 yrs. 7 mos.
Temporal	9	0	9 (100%)	2 yrs. 4 mos.
Frontoparietal	5	2 (40%)	3 (60%)	6 yrs. 5 mos.

TABLE 17

Survival by age group—astrocytoma diffusum (including 2 biopsies)

Age Group (Years)	No. of Patients	No. Alive	No. Dead	Average Survival
1–10	1	—	1 (100%)	8 mos.
11–20	0	—	—	—
21–30	4	2 (50%) including 1 biopsy only	2 (50%)	6 yrs. 1 mo.
31–40	13	3 (23%)	10 (77%) including 1 biopsy	4 yrs.
41–60	4	0	4 (100%)	1 yr. 2 mos.

TABLE 18

Mortality after surgery—astrocytoma diffusum (17 cases)

No. of Deaths	4	3	4	3				1	1	1
Per Cent	23%	18%	23%	18%				6%	6%	6%
No. of Years after Surgery	0–1	1–2	2–3	3–4	4–5	5–6	6–7	7–8	8–9	9–10

TABLE 19

Morbidity in 5 cases of astrocytoma diffusum—patients now living

Case No.	Present Condition of Patient	A.S.E. Code*	Survival
Residual Disability but Self-Supporting—4 cases			
37	Patient had second removal 1¼ yrs. after first. No signs of recurrence now; 2 seizures last year	434	9 yrs. 9 mos.
49	Occasional minor seizures; no signs of disease	434	7 yrs. 10 mos.
90	Difficulty with speech; weakness right hand and foot	334	5 yrs. 3 mos.
106	Slight difficulty with speech	434	4 yrs. 6 mos.
Residual Disability and not Self-Supporting—1 case			
107	Mild seizures 2–3 a week for 6 mos.; hemiparesis; partly cares for self; doesn't work	220	4 yrs. 3 mos.

* A=anatomical, S=symptomatic, E=economical: 4 is a perfect score; 0 is complete failure. A perfect score means there is no major organic symptom or sign and the patient can work.

TABLE 20

Morbidity in 17 cases of astrocytoma diffusum—patients now dead

No. of Patients	Survival	Postoperative Condition
5	2 yrs. or more	Led useful lives
5	2 yrs. or more	Unknown
7	Unknown	Unknown

3. GEMISTOCYTIC ASTROCYTOMA

There were 27 cases in this category. The average age of the patients at admission was 40 years (Table 21) and 38 years at onset of symptoms. Sixteen were males, and 11 were females.

The average duration of symptoms before operation was 25 months. In contradistinction to the piloid and diffuse groups, headache occurred as a presenting symptom more frequently than seizures. Eighteen (67 per cent) patients had headache and 17 (65 per cent) had seizures. Papilloedema occurred in 15 (60 per cent), and hemiparesis was more common in this group.

The location of the tumours and the relationship of seizures to the region affected are shown in Tables 22 and 23.

Macroscopic cysts were found in 7 (26 per cent) instances. Only 2 (31 per cent) of the 7 patients had convulsions, and again there seems to be no reason to relate the presence of a cyst with the presence of convulsions, which were present in 65 per cent of all cases in this subgroup.

Twenty-seven surgical specimens were examined histologically. Microscopic calcium was not found in any specimen although it had been seen by roentgen ray in 4 (15 per cent). Three specimens showed some tendency towards malignancy while 9 (33 per cent) showed occasional mitoses.

TABLE 21

Age incidence—gemistocytic astrocytoma (27 cases)

No. of Cases	1	0	5	9	6	2	4
Age (years)	1–10	11–20	21–30	31–40	41–50	51–60	61–70

TABLE 22

Location—gemistocytic astrocytoma (27 cases)

Location	No. of Cases	Per Cent
Frontal	10	37%
Parietotemporal	5	18%
Parietal	5	18%
Temporal	4	15%
Frontoparietal	3	12%
Right-sided	12	
Left-sided	14	
Bilateral	1	

Operative Summary. Surgical procedures were carried out on 27 patients. Two patients had a further removal of tumour 3 years, and 1 year and 10 months after the initial excision and survived a further 1 year, and 1 month respectively after their second operation. The latter patient was one in whom a complete removal appeared to have been carried out on the first occasion.

Thirty-one operative procedures were carried out on 27 patients. There were 3 postoperative deaths, a case mortality of 11 per cent.

In 21 cases removal of the tumour (1 crossed the midline) was incomplete, and in 6 cases it was described as complete.

The average survival of the 6 patients with tumours that were thought to be completely removed was 3 years and 10 months (including 3 patients who are still alive), while the survival of the remaining 18 with known incomplete removals was 2 years, 7 months (including 2 patients who are still alive). Survival and morbidity are shown in Tables 24–30 inclusive.

TABLE 23

Relation of seizures to region affected—gemistocytic astrocytoma

Region	No. of Cases	Major Seizures	Minor Seizures	Total Seizures	Percentage
Frontal	10	6	1	7	70%
Parietal	5	2	1	3	60%
Temporal	4	3	0	3	75%
Parietotemporal	5	2	0	2	40%
Frontoparietal	3	2	0	2	66%

TABLE 24

Survival after surgical removal—gemistocytic astrocytoma

Dead—19 cases

Case No.	Survival	Case No.	Survival	Case No.	Survival
44	8 yrs.	78*	1 yr. 10 mos.	60*	6 mos.
25	6 yrs.	9*	1 yr. 8 mos.	24	4 mos.
67*	4 yrs.	1*	1 yr. 6 mos.	15	3 mos.
81*	3 yrs. 4 mos.	48	1 yr. 4 mos.	28*	3 mos.
26	3 yrs. 2 mos.	57*	1 yr.	62	3 mos.
77	3 yrs. 1 mo.	32*	10 mos.		
91*	2 yrs. 7 mos.	4*	8 mos.		

Alive—5 cases

Case No.	Survival	A.S.E.†	Case No.	Survival	A.S.E.†
55	7 yrs. 6 mos.	331	102	5 yrs.	220
85	6 yrs. 6 mos.	222	113*	4 yrs. 5 mos.	334
65*	6 yrs.	440			

* Received roentgen-ray therapy.

† A = anatomical, S = symptomatic, E = economical: 4 is a perfect score; 0 is complete failure. A perfect score means there is no major organic symptom or sign and the patient can work.

4. THIRD VENTRICLE ASTROCYTOMAS

There were 5 cases in this group. They are discussed separately because of the unique situation of the tumours and the problems associated with their operative removal.

The patients were aged 17, 37, 39, 12 and 4 years respectively, with an average age of 22 years. Three were males, and 2 were females. The average pre-operative duration of symptoms was 17 months but if the last case is excluded, that of a child with a large head and a decompression provided thereby, the duration is reduced to 10 months.

TABLE 25

Comparative survivals—gemistocytic astrocytoma (24 cases)

1. Average survival regardless of treatment (5 living, 19 dead)—2 years 11 months.
2. Average survival after surgery alone (3 living, 9 dead)—3 years 6 months.
3. Average survival after surgery and roentgen-ray therapy (2 living, 10 dead)—2 years 3 months.
4. Average survival of 7 patients with macroscopic cysts (2 living, 5 dead)—3 years 9 months.
5. Average survival of 4 patients with macroscopic calcium (2 living, 2 dead)—5 years.
6. Average survival of 9 patients with tumours showing mitotic figures (1 living, 8 dead)—2 years 2 months.
7. Average survival of 3 patients with tumours showing malignant tendency (1 living, 2 dead)—3 years 7 months.
8. Five-year survival: 6 patients (2 dead, 4 living) out of 24 (25%). If 1 patient who is likely to survive 5 years is included, probable survival would be 29%.

TABLE 26

Survival by age group—gemistocytic astrocytoma (24 cases)

Age Group (Years)	No. of Patients	No. Alive	No. Dead	Average Survival
0–10	1	—	1	8 yrs.
11–20	0	—	—	—
21–30	4	1 (25%)	3 (75%)	3 yrs. 2 mos.
31–40	9	2 (22%)	7 (78%)	2 yrs. 9 mos.
41–50	5	1 (20%)	4 (80%)	1 yr. 7 mos.
51–60	2	1 (50%)	1 (50%)	6 yrs. 9 mos.
61–65	3	0	3 (100%)	3 mos.

TABLE 27

Mortality after surgery—gemistocytic astrocytoma (19 cases)

No. of Deaths	7	5	1	3	1		1		1	
Per Cent	37%	26%	5%	15%	5%		5%		5%	
No. of Years after Surgery	0–1	1–2	2–3	3–4	4–5	5–6	6–7	7–8	8–9	9–10

TABLE 28

Average survival relative to site of tumour—gemistocytic astrocytoma

Region	No. Patients	No. Living	No. Dead	Average Survival
Frontal	9	1 (11%)	8 (89%)	2 yrs. 7 mos.
Temporal	3	1 (33%)	2 (66%)	3 yrs. 4 mos.
Parietal	5	1 (20%)	4 (80%)	3 yrs. 10 mos.
Parietotemporal	5	1 (20%)	4 (80%)	2 yrs. 4 mos.
Frontoparietal	2	1 (50%)	1 (50%)	3 yrs. 4 mos.

TABLE 29

Morbidity in 5 cases of gemistocytic astrocytoma—patients now living

Case No.	Present Condition of Patient	A.S.E. Code*	Survival
113	Mild hemiparesis	334	4 yrs. 5 mos.
85	Hemiparesis. Occasional seizures	222	6 yrs. 6 mos.
55	Slight speech difficulty. Hemianopic. Had one seizure; may have recurrence	331	7 yrs. 6 mos.
65	No symptoms. Slight personality change. Doesn't work	440	6 yrs.
102	Blind. Convulsions recently	220	5 yrs.

* A = anatomical, S = symptomatic, E = economical: 4 is a perfect score; 0 is complete failure. A perfect score means there is no major organic symptom or sign and the patient can work.

TABLE 30

Morbidity in 19 cases of gemistocytic astrocytoma—patients now dead

No. of Patients	Survival	Postoperative Condition
3	2 yrs. or more	Led useful lives for several years
4	2 yrs. or more	Unknown
12	Unknown	Unknown

Signs of increased intracranial pressure were present in all cases. In the first 4 cases there was papilloedema while in the fifth case there was a "crack-pot sound" and an enlarged head. Two patients had had convulsions.

All 5 patients were operated upon through frontal bone flaps and an attempt was made to remove the tumour either through the corpus callosum or through the foramen of Monro.

Four patients died in the immediate postoperative period. The fifth, a boy aged 17, survived his first operation and continued asymptomatically for 6 years. During the War he worked in his father's factory. He was given a course of roentgen-ray therapy totalling 4,000 r (tumour dose). Symptoms of ventricular obstruction returning, this patient was re-explored 7 years later and a further removal was carried out. He died 6 days later. At autopsy the tumour was found to have involved the hypothalamus, 3rd ventricle and interpeduncular regions.

Histological specimens were re-examined in all of these cases. The tumours have been classified as piloid astrocytomas. No calcium was seen. There were no mitoses but a cyst was present in one.

5. OPTIC NERVE ASTROCYTOMAS

There were 3 patients in this group. Their ages were 9 years, 5 years, and 21 months. Two were female, and 1 was male. The average pre-operative duration of symptoms was 23 months.

Two patients were admitted with signs of increased intracranial pressure, while the youngest had exophthalmos. Bilateral optic atrophy was found in 2, while papilloedema was present in the third. No patient had suffered seizures.

Radical removal of a tumour of the optic chiasm and hypothalamus was undertaken in 1 patient, a girl of 9 years. She subsequently lived a further 9 years but was blind and hemiparetic at death. A large removal was made in the other 2 patients, both of whom died the day following operation.

The 3 surgical specimens were classed as piloid astrocytoma. One contained microscopic calcium and a cyst while another showed mitoses.

6. BORDERLINE ASTROCYTOMAS

This group consists of 5 cases in which, histologically, the tumour was predominantly an astrocytoma, but in each case it presented some areas

TABLE 31

Location of borderline astrocytomas

Location		No. of Cases
Frontal		2
Occipital		1
Corpus callosum and lateral ventricle		1
Parietal		1
Right-sided	3	
Left-sided	2	

that were by no means typical of this lesion, and yet it was found to be unclassifiable in any other way. These tumours have been variously subclassified as "malignant piloid" (2 cases), "borderline astrocytoma" (2 cases), and "malignant astrocytoma" (1 case).

The average age of the 5 patients was 51 years; the oldest was 75, and the youngest 22 years. All 5 were males.

The average duration of symptoms in this group was 16 months, the longest being 3 years with convulsions, and the shortest being 3 months with increasing paralysis. Three patients had suffered from headache, 3 from seizures and 3 had papilloedema.

The general location of the tumours and the relationship of seizures to location are shown in Tables 31 and 32.

These tumours were not grossly cystic. Calcium was present in 3 cases and was seen in the roentgenogram in another case in which it was not histologically visible.

Operative Summary. One patient (No. 75), a man of 75 years, had a biopsy and decompression only. He had had symptoms for only 3 months, and in view of his age it was felt that operative removal was not warranted. He died 8 months later. Four patients had a surgical removal (Nos. 30, 34, 93, 94). One patient (No. 30), aged 58, had a second operation $5\frac{1}{2}$ years after his first operation followed by a course of deep roentgen-ray therapy (6,500 r). When last heard from, he was alive and well—10 years and 3 months after his first operation, and without signs of muscle weakness. This tumour was situated in the right frontal region. Six operative procedures were performed on 5 patients. There was no operative mortality.

Follow-up data are summarized in Table 33.

TABLE 32

Relation of seizures to location—borderline astrocytomas

Region	No. of Cases
Frontal	1
Corpus callosum	1
Parietal	1

TABLE 33

Survival after surgery—borderline astrocytomas

Alive			
Case No.		Survival	A.S.E.†
30*		10 yrs. 3 mos.	332
Dead			
Case No.	Survival	Case No.	Survival
93*	5 yrs.	94*	7 mos.
34	1 yr. 6 mos.	75	8 mos. (biopsy and decompression only)

Average survival of all patients regardless of treatment—3 yrs. 7 mos. (1 living, 4 dead)

* Received roentgen-ray therapy.

† A=anatomical, S=symptomatic, E=economical: 4 is a perfect score; 0 is complete failure. A perfect score means there is no major organic symptom or sign and the patient can work.

7. COMPARATIVE STUDY OF VARIOUS SUBGROUPS

Table 34 shows that the four subgroups of supratentorial astrocytomas affect patients at an increasing age in the order given, and that there is a concomitant increase in malignancy.

Table 35 indicates that patients with diffuse astrocytomas have the highest incidence of seizures and the lowest of papilloedema.

It is probable that the higher incidence of seizures occurring with the diffuse astrocytomas is related as much to the site and rate of the growth as to its specific characteristics. Seizures are least common when the tumour occupies the deep or posterior half of the hemisphere. These two areas were not involved in any of the 23 cases of diffuse astrocytomas recorded here. Seizures are less common in cases of the more malignant gemistocytic astro-

TABLE 34

Supratentorial astrocytomas. Summary of age incidence, duration of symptoms and postoperative survival

Type	No. of Patients Followed	Average Age (Years)	Duration of Symptoms (Months)	Still Alive %	Dead %	All Dead—Dying Within 1 Year %	Average Survival (All Cases)
Piloid	38*	33½	49	45%	55%	19%	4 yrs.
Diffuse	22	35	41	23%	77%	23%	3 yrs. 9 mos.
Gemistocytic	24	40	25	21%	79%	37%	2 yrs. 11 mos.
Borderline	5	51	16	20%	80%	50%	3 yrs. 7 mos.

* Biopsies excluded.

cytomas. It is a common observation that the more malignant tumours are more prone to produce paralyses and less likely to produce seizures, which may be because of a shorter life history.

Four patients (3 gemistocytic and 1 diffuse) gave a history of previous head injury. In 2 it was related to the onset of symptoms. This seemed to be quite fortuitous.

Symptoms were associated with pregnancy in 3 cases. In 1, symptoms

TABLE 35

Symptoms—supratentorial astrocytomas

Group	Seizures	Headache	Seizures Only	Papilloedema
Diffuse	82%	39%	43%	26%
Piloid	74%	55%	28%	43%
Gemistocytic	65%	67%	11%	60%
Borderline	60%	60%	—	60%

dated from the commencement of pregnancy, and in a second from the time of delivery. Both tumours were diffuse astrocytomas. A third patient had been investigated at the age of 5 years and roentgenograms showed a calcified mass in the midline of the skull anteriorly. Operation was refused. When the patient was aged 26, and when 5 months pregnant, headaches and vomiting developed, and 1 week later she became hemiparetic. She was admitted comatose with decerebrate rigidity and died after operation. She had a piloid astrocytoma of the interventricular region.

One of our patients had an undisturbed pregnancy and delivery 5 years after the removal of a left temporal astrocytoma, and was asymptomatic at her last examination 5 years 11 months after operation.

Astrocytoma of the brain may occur at any age. The peak incidence, however, lies between 20 and 45 years.

The majority of piloid astrocytomas occurs in the frontal lobe (40 per cent), the temporal lobe being the next most frequently affected. The frontal and the temporal regions contained 78 per cent of the diffuse astrocytomas and there was none posteriorly, and there was none of the gemistocytic type in the occipital region.

Survival was longest in those cases of piloid astrocytomas occurring in the temporal region, but this region carried the poorest prognosis for the gemistocytic type. The frontal region carried almost the poorest prognosis for the gemistocytic type, and only a fair prognosis for the diffuse type. It seems likely that factors other than the site of growth have a greater bearing upon prognosis than the actual locality from which the tumour was removed since both long and short survivals were obtained from all regions.

Macroscopic cysts were present most frequently in the piloid group. There was no significant difference between the survival times of patients who had macroscopic cysts and those who had none, and they cannot be taken

as an indication of benignity. Similarly, calcium was found most frequently in the piloid type. There was a slight but insignificant increase in survival of those patients as opposed to the whole group but, on the other hand, the few such patients in the diffuse group had a much shorter survival time than average, so that it is probably of no significance.

The presence of mitotic figures is by no means a definite indication of poorer prognosis. The survival time in 7 cases of piloid astrocytomas with mitoses was slightly above average, as it was in cases of diffuse tumours with mitoses. On the other hand, in cases of gemistocytic astrocytomas with mitotic figures, the patients lived 1 year and 6 months less than the average of the whole group. A tendency toward malignancy was noted in 4 piloid cases and 3 gemistocytic cases. There was no significant shortening of the survival time in either of these two groups.

There is not a great variation in the average survival time of patients in the three major groups who are still alive (Table 36). It is likely, however, that with longer follow-up the more malignant types will drop out.

TABLE 36

Average survival of patients in each group of astrocytomas

Group	Dead			Alive		
	No. of Cases	Per Cent	Average Survival	No. of Cases	Per Cent	Average Survival
Piloid	21	55%	36 mos.	17	45%	65 mos.
Diffuse	17	77%	36 mos.	5	23%	76 mos.
Gemistocytic	19	79%	26 mos.	5	21%	70 mos.
Borderline	4	80%	23 mos.	1	20%	123 mos.

A considerable problem is posed in the treatment of patients with diffuse astrocytomas. One (No. 37) treated by surgical removal on 2 occasions as well as by roentgen-ray therapy survived, and is still alive without evidence of recurrence, 9 years and 9 months after her first operation. Another (No. 23) had a craniotomy with a large biopsy, but no roentgen-ray therapy. He survived for 9 years. Case 49 had a biopsy of an infiltrating lesion in the precentral gyrus, a course of roentgen-ray therapy and is still alive, 7 years and 10 months later. Other patients in whom large removals were performed lived for periods considerably shorter than these, some with roentgen-ray therapy as well, some without.

In the cases of piloid astrocytomas, there is very little difference in the average survival of those who had an incomplete and those who were thought to have had a complete removal (Table 37). With longer follow-up there is no doubt that the balance will swing in favour of the so-called complete removals.

In the cases of diffuse astrocytomas the average survival after incomplete

TABLE 37

Average survival after surgical removal—supratentorial astrocytomas

Group	Complete Removal	Incomplete Removal
Piloid	3 yrs. 10 mos. (10 dead, 6 living)	4 yrs. 2 mos. (11 dead, 11 living)
Diffuse	1 yr. 10 mos. (5 dead, 0 living)	3 yrs. 7 mos. (12 dead, 3 living)
Gemistocytic	3 yrs. 10 mos. (3 dead, 3 living)	2 yrs. 7 mos. (16 dead, 2 living)

removal was considerably longer than that after so-called complete removal, while in the gemistocytic group, the average survival after complete removal was 1 year and 3 months longer than that after the incomplete. The results of course may be affected by cautious reporting by the surgeon. From other longer follow-up studies it is known that patients in the piloid group may survive indefinitely after surgery. The writers prefer complete removal whenever possible. A 5-year survey of all patients with follow-up reports after surgery is given in Table 38.

Of 38 patients with piloid astrocytoma followed after operation (excluding biopsy), 17 were alive at the time of writing and 14 were self-supporting more than 4 years after operation. Of 22 patients with diffuse astrocytoma (including 2 with biopsy), 5 were alive and 4 (1 with biopsy) were self-supporting more than $4\frac{1}{2}$ years after operation. Of 24 patients with gemistocytic astrocytoma, 5 were living and 1 was self-supporting more than $4\frac{5}{12}$ years after operation. Of 21 deceased patients in the piloid group (excluding biopsy) 12 survived more than 2 years, and 7 were self-supporting before death. Of 17 deceased in the diffuse group (including 1 biopsy only, 10 survived 2 years or more; interim follow-up incomplete) 5 are known to have led useful lives for several years. Of 19 deceased in the gemistocytic group (only 7 survived 2 years or more; interim follow-up incomplete) 3 are known to have led useful lives.

Of the 84 patients with astrocytoma of the cerebrum (excluding the 3rd

TABLE 38

Five-year survey of 97 patients with supratentorial astrocytomas who survived surgery

Group	No. of Patients Followed	No. Who Survived 5 Years	Percentage
Piloid	38	14	37%
Diffusum	22 (includes 2 biopsies)	6	27%
Gemistocytic	24	6	25%
3rd ventricle	5	1	20%
Optic nerve	3	1	33%
Borderline	5	2	40%
	97	30	31%

ventricle) 27 (32 per cent) are alive and 19 (22 per cent) are self-supporting.

Laboratory Studies. The diagnostic value of certain laboratory techniques is shown in Tables 39–43 inclusive.

The maximal and minimal readings for the protein content of the spinal fluid and the ventricular fluid for the various subgroups of astrocytoma are listed in Table 39.

TABLE 39

Cerebrospinal fluid studies

Protein Content		Piloid	Diffuse	Gemistocytic	Borderline	3rd Ventricle	Optic Nerve
Spinal fluid	Max.	250	74	100	47	160	40
	Min.	10	13	18	43	—	20
Ventricular fluid	Max.	67	69	77	64	10	—
	Min.	10	10	63	—	—	—

Martin, Webster and Gurdjian[3] found that when posterior fossa and midline basilar lesions were excluded from their series of 224 electroencephalograms 59 per cent were accurately localized, and when malignant meningiomas and rapidly growing gliomas were considered alone localization was correct in 66 per cent. Renfrew, Cameron and Haggar[4] stated, on the basis of 56 cases, that 94.5 per cent of their tumour patients had an abnormal electroencephalogram and that the electroencephalogram can lateralize 74.5 per cent of tumours and locate 65.4 per cent. These figures are in keeping with the findings listed in Table 40.

Plain roentgenograms of the skull were done in 93 cases (Table 41).

Pneumoencephalography was performed 50 times on 47 patients (Table 42).

Ventriculography was performed on 62 patients (Table 43). Localization was correct in 88 per cent of cases and adequate in the remaining 12 per cent.

TABLE 40

Electroencephalographic studies

Group	Correctly Localizing	Abnormal, not Localizing	Incorrect	Normal	Total Cases
Piloid	19	8	3	1	31
Gemistocytic	10	3	3	—	16
Diffuse	11	6	1	—	18
Borderline	1	1	—	—	2
3rd ventricle	1	1	—	—	2
	—	—	—	—	—
	42 (61%)	19 (27%)	7 (10.5%)	1 (1.5%)	69

TABLE 41

Roentgenological studies

Group	Total No.	Normal	Calcium Present	Pineal Shift	Intra-cranial Pressure	Other Correct Findings	Localiza-tion Incorrect
Piloid	42	13	9	7	10	4	3
Gemistocytic	22	6	4	6	10	—	—
Diffuse	17	10	1	4	2	1	—
3rd ventricle	5	—	—	—	5	—	—
Borderline	4	—	2	—	1	1	—
Optic nerve	3	1	1	—	—	1	—
	—	—	—	—	—	—	—
	93	30	17	17	28	7	3

TABLE 42

Pneumoencephalographic studies

Group	Total No.	Correct	Interpre-tation Impossible	Adequately Localizing	Normal Filling
Piloid	18 (17 pts.)	16	2	—	—
Gemistocytic	8	7	—	1	—
Diffuse	19 (17 pts.)	16	3	—	—
Optic nerve	2	—	1	—	1
Borderline	3	3	—	—	—
	—	—	—	—	—
	50 (47 pts.)	42	6	1	1

TABLE 43

Ventriculographic studies

Group	Total	Localization Correct	Localization Adequate
Piloid	30	27	3
Gemistocytic	17	13	4
Diffuse	7	7	—
3rd ventricle	5	5	—
Optic nerve	2	2	—
Borderline	1	1	—
	—	—	—
	62	55	7

II. ASTROCYTOMAS OF THE POSTERIOR FOSSA

There were 44 astrocytomas located in the posterior fossa. They have been subdivided according to their gross site of origin as shown in Table 44.

The average age at the time of admission of patients with midline tumours was 15½ years, and of those with hemispheral tumours, 12 years. The average age of all patients except those with pontine tumours, which will be

TABLE 44

Location of astrocytomas of posterior fossa

Midline structures—vermis and 4th ventricle	24
Cerebellar hemisphere	15
Site of origin uncertain	2
Pons	3

considered separately, was 13 years, 11 months. There were 27 males and 14 females.

The average duration of symptoms was 7 months in the midline cases and 17 months in the hemispheral cases. In the 24 midline cases, symptoms of increased intracranial pressure appeared before or at the same time as those of cerebellar dysfunction in 18 instances. Some sign of cerebellar damage was elicitable in every case but one. In the 15 hemispheral cases symptoms of raised intracranial pressure appeared first in 8 cases. In every case signs of cerebellar damage were elicitable.

Headache, vomiting, papilloedema, and unsteadiness of gait occurred about equally in the two groups. Papilloedema occurred in 80 per cent and optic atrophy in 4–7 per cent of cases. Double vision was twice as common in the hemispheral cases. Dizziness was more than four times as common in the midline cases. Nystagmus was more common in midline cases. Hyporeflexia was more than three times as frequent in the hemispheral cases. Paresis of the 7th nerve was five times more common in the hemispheral cases. Enlargement of the head was four times as frequent in midline cases; however, examination of the age incidence suggests that this is probably attributable to chance selection.

All 41 patients underwent surgical intervention. There were 4 postoperative deaths, a case mortality of 9.7 per cent.

Five patients had further surgical procedures as follows:

No. 1. Re-operation after $7\frac{1}{2}$ years for recurrence of symptoms, but no tumour was present. Cysts were excised from both hemispheres.

Nos. 18, 25, 26. Plastic repair of subcutaneous cerebrospinal fluid cysts.

No. 30. Ventriculoperitoneal shunt performed 1 month after removal of the tumour. The patient then commenced to improve.

Forty-one surgical specimens were re-examined. All were classified as piloid astrocytoma. Two of these (Cases 27 and 29) were considered to be slightly more malignant than the others but not sufficiently so as to warrant their being placed in a separate category.

Macroscopic cysts were present in 18 (75 per cent) of the 24 midline cases, and in 14 (93 per cent) of the 15 hemispheral cases. Three cysts were present in 1 patient who had been operated upon previously, elsewhere, and in whom the site of origin of the tumour was uncertain (No. 4).

Microscopic calcium was seen in 5 hemispheral cases (Nos. 5, 28, 31, 33 and 35) and in 2 midline cases (Nos. 10 and 41). It was demonstrated by

roentgen ray in 2 cases (Nos. 5 and 10). Mitoses were seen in 2 midline tumours (Cases 8 and 32), 1 cystic and 1 solid, and in 1 hemispheral tumour without cyst (Case 2).

1. CEREBELLAR ASTROCYTOMAS

Midline Cerebellar. There were 24 cases in this group. Four (16.66 per cent) patients died in the immediate postoperative period. They had cystic tumours with extensive involvement. Another patient (No. 10) has been lost to follow-up.

Thus 19 patients were available for study of postoperative results. Four of these died within 2 years of surgery (Nos. 17, 25, 26, 29). One had a cystic tumour, and 3 had solid tumours. In the case of the cystic tumour, removal was believed to have been complete. In the 3 patients with solid tumours, removal was known to have been incomplete in 2 of them, and although a complete removal was believed to have been effected in the third, the tumour was somewhat more malignant than usual.

Fifteen patients are alive, their postoperative periods ranging from $4\frac{1}{2}$ to 14 years (Table 45). Eleven are in good health and work or attend school. Four carry a disability and 3 of these do not work. No. 40 is blind and

TABLE 45

Longevity after surgical removal—midline cerebellar astrocytoma (24 cases)

Died in hospital	4
Lost to follow-up	1
Died under 2 years	4
Alive	15

Dead—4 cases

Case No.	Survival	Case No.	Survival
26	2 yrs.	17	5 mos.
25	1 yr.	29	5 mos.

Alive—15 cases

Case No.	Survival	A.S.E.†	Case No.	Survival	A.S.E.†
2	14 yrs. 2 mos.	330	39*	7 yrs. 7 mos.	444
8*	12 yrs. 5 mos.	444	43	7 yrs. 6 mos.	334
6	12 yrs.	444	21	7 yrs.	444
9*	10 yrs.	344	23	6 yrs.	232
18	10 yrs.	444	27	6 yrs.	320
40	8 yrs.	220	30	4 yrs. 9 mos.	333
37	8 yrs. 7 mos.	334	32	4 yrs. 6 mos.	444
41	7 yrs. 10 mos.	444			

* Received roentgen-ray therapy.

† A = anatomical, S = symptomatic, E = economical: 4 is a perfect score; 0 is complete failure. A perfect score means there is no major organic symptom or sign and the patient can work.

hemiparetic, with some emotional change 8 years after operation. No. 23 is alive 6 years after operation, but is blind. He is, however, of good morale and is self-supporting. No. 27 has some neurological deficit 6 years after operation, but no signs of recurrence. He could work, but the situation of the current labour market prevents his obtaining employment. No. 2 is alive and well 14 years and 2 months after operation but does not work, though he well could, because his family treats him as an invalid. His vision is poor.

A summary of the follow-up and morbidity data is given in Tables 45 and 46.

Cerebellar Hemispheral. There were 15 cases in this group. There were no postoperative deaths. Three patients have been lost to follow-up. Twelve patients are available for study and are alive and well, their longevity after surgery ranging from $4\frac{3}{4}$ to $13\frac{1}{2}$ years.

Follow-up and morbidity data are summarized in Tables 47 and 48.

TABLE 46

Morbidity in 15 cases of midline cerebellar astrocytoma—patients now living

Morbidity	Case No.	A.S.E. Code*	Result
Normal	8, 6, 18, 41, 39, 21, 32	444	Working
Hemiparesis and blind	40	220	Not working
Cerebellar signs	30	333	Working
	9	344	Working
	27	320	Could work
VIIth nerve palsy	37	334	Working
Blind in 1 eye and right cerebellar signs	2	330	Could work
Blind in both eyes	23	232	Working
Tires easily	43	334	Working

* A=anatomical, S=symptomatic, E=economical: 4 is a perfect score; 0 is complete failure. A perfect score means there is no major organic symptom or sign and the patient can work.

TABLE 47

Longevity after surgical removal—cerebellar hemispheral astrocytoma (12 cases)

Case No.	Survival	A.S.E.†	Case No.	Survival	A.S.E.†
1	13 yrs. 6 mos.	334	19	9 yrs. 3 mos.	333
7	12 yrs. 6 mos.	444	35	8 yrs. 6 mos.	234
5	12 yrs.	232	24	6 yrs.	234
12	10 yrs.	343	33	5 yrs. 6 mos.	444
14	9 yrs. 6 mos.	334	28*	4 yrs. 9 mos.	444
13*	9 yrs. 6 mos.	334	31	4 yrs. 9 mos.	433

* Received roentgen-ray therapy.

† A=anatomical, S=symptomatic, E=economical: 4 is a perfect score; 0 is complete failure. A perfect score means there is no major organic symptom or sign and the patient can work.

TABLE 48

Morbidity in 12 cases of cerebellar hemispheral astrocytoma—patients now living

Morbidity	Case No.	A.S.E. Code*
Physically and mentally normal	7, 24, 33, 28	444
Blind	35	234
Poor vision and ataxia	1	334
Poor vision	5	232
Labile personality	31	433
Slight weakness 1 side	12	343
Slight weakness 1 side and diminished Vth nerve	13	334
Strabismus and headaches	19	333
Poor vision 1 eye	14	334

* A=anatomical, S=symptomatic, E=economical: 4 is a perfect score; 0 is complete failure. A perfect score means there is no major organic symptom or sign and the patient can work.

Site of Origin Uncertain. There were 2 cerebellar astrocytomas in this category. There were no postoperative deaths. Case 3 has survived 13 years and 10 months since operation and is free of signs, with an A.S.E. code of 444. Case 4 has survived 13 years since second operation performed here and 20 years since primary operation performed elsewhere. Muscle recession was for strabismus. A.S.E. code is 344.

Diagnostic Localization. Plain roentgenograms of the skull were taken on 31 patients. Signs of increased intracranial pressure were present in 24 (77 per cent). Calcium was present in 2 (6.5 per cent). Seven patients had a normal roentgenogram (22 per cent). There was an unrelated abnormality in 2 (6.5 per cent).

Pneumoencephalography was performed on 5 patients, twice on 1 patient, three times on another. It was adequately localizing in 1 case only, when the tumour was located in the hemisphere.

Ventriculography was performed on 37 patients and gave a correct localization in every case. The cerebrospinal fluid protein was estimated in 7 of our cases and levels ranged from 35–178 mg. per cent. The ventricular fluid protein was examined in 15 cases, and the levels ranged from 10–49 mg. per cent.

Electroencephalography was performed on 10 patients. Localization to the posterior fossa, or better, was obtained in 5 (50 per cent) cases. An abnormal but non-localizing record was obtained in 3 (30 per cent) cases, and an incorrect localization in 2 (20 per cent) cases. In 41 cases localization was made:

a) On clinical grounds alone	2 cases (5 per cent)
b) By plain roentgenograms alone	1 case (2.5 per cent)
c) By pneumoencephalography	1 case (2.5 per cent)
d) By ventriculography	37 cases (90 per cent)

2. ASTROCYTOMA OF THE PONS

Of 3 patients, 2 were male and 1 was female. The average age at the time of admission was 6 years. Symptoms had been present for 3 years, 4 months, and 1 month respectively. In the first case these had consisted of headache and increased intracranial pressure, while in the other 2 cases symptoms of paralysis were more marked than those of ventricular obstruction. Signs of involvement of the pyramidal tracts and paresis of one or more of the cranial nerves were present in every case. Nystagmus was present in all 3 cases and papilloedema in 1 patient.

Surgical exploration was performed on all 3 patients. No removal of tumour was possible. All died within 1 month of the operation.

The tumours were all classified as piloid astrocytoma. There were no cysts in any of the specimens and mitoses were seen in only 1 tumour. There was no microscopic calcium.

Roentgenogram of the skull was normal in 2 cases. In 1 patient (No. 20) who had had headaches for 3 years, there was evidence of increased intracranial pressure. There was no abnormal calcification seen in any of the roentgenograms.

In 1 case (No. 22), neither pneumoencephalography nor ventriculography definitely localized the lesion to the pons, though its position in the posterior fossa was confirmed. In Case 15, localization was made clinically, and in Case 20 it was made by ventriculography.

Cerebrospinal fluid protein varied from 58 to 244 mg. per cent in the spinal fluid and from 15 to 54 mg. per cent in the ventricular fluid.

Electroencephalography was performed in 1 case (No. 22). This showed a deep-seated lesion without evidence of lateralization.

3. DISCUSSION OF POSTERIOR FOSSA ASTROCYTOMAS

Apart from minor differences in symptomatology and physical signs, there is nothing that would enable the clinician to differentiate a midline cerebellar lesion from one in the cerebellar hemisphere. Ataxia, especially truncal ataxia, is reputed to be associated with midline lesions but was associated about equally with tumours of the cerebellar hemispheres.

Tumours of the pons produced a higher incidence of pyramidal tract signs. If 3 cases can be considered significant, nystagmus was present in 100 per cent of pontine cases as against 47 per cent of cerebellar cases. Symptoms were present on the average for a longer time in patients with a hemispheral lesion than in patients with a midline lesion.

The pontine astrocytomas occurred at a slightly earlier age (average 6 years) than did the others (average 15½ and 12 years), and the symptoms were somewhat more bizarre. Papilloedema, which was present in 78 per cent of the patients with cerebellar tumours, occurred in only 33 per cent of the 3 with pontine tumours.

Histologically the tumours did not vary greatly in their characteristics. Cysts were present slightly more frequently in the hemispheral astrocytomas, and there was none in the pontine tumours.

From the surgical standpoint tumours of the cerebellar hemisphere are a better risk, both at the time of operation and over the years, than the midline tumours. There were 4 operative deaths, all occurring in cases of midline cystic astrocytomas. There were 4 deaths during the following 2 years, all occurring in midline cases. Three of the tumours in these cases were noncystic. There were no operative deaths among the hemispheral cases (3 have been lost to follow-up), nor have any of the patients died since discharge from hospital. This also applies to the 2 cerebellar cases of uncertain origin listed separately.

In this series 3 patients with noncystic midline tumours, of a total of 6, are alive more than 10 years after operation. The average duration of symptoms of the 3 patients who lived was 10 months, while the duration of symptoms of those who died was 5 months.

At surgery, a complete removal was believed to have been performed in 70 per cent of the midline cases and in 73 per cent of the hemispheral cases. Of the 7 patients with midline tumours in whom an incomplete removal was performed, 4 (57 per cent) are still alive and well $12\frac{1}{4}$, 12, $8\frac{1}{2}$ and $4\frac{3}{4}$ years later. Two (28 per cent) died within a year, and 1 (15 per cent) died at the time of surgery. Of 3 patients with hemispheral tumours so treated and followed, all are still alive $9\frac{1}{2}$, $9\frac{1}{2}$ and 5 years later (100 per cent). On the other hand, a complete removal was thought to have been performed in 16 midline and 8 hemispheral cases. Of the former group, 4 (25 per cent) patients died postoperatively, and 2 (12 per cent) died within 2 years. The remaining 10 (63 per cent) are well for periods of 4 to 14 years. Of the 8 patients who had complete removal of hemispheral tumours, all are well for periods of $4\frac{3}{4}$ to $13\frac{1}{2}$ years (100 per cent).

All 3 patients with pontine lesions died either at surgery, or postoperatively. Surgery should be directed towards the relief of ventricular obstruction in these cases, rather than to a fruitless attack upon the tumour, provided the diagnosis can be made in advance.

A total of 41 patients with cerebellar astrocytoma have been treated by surgical removal of the tumour. Four patients died at surgery. Five underwent further surgery, only 1 of them for recurrence of symptoms which turned out to have been caused by cysts. The case mortality was 9.7 per cent. Twenty-nine patients are known to be living (4 have been lost to follow-up, but 1 of these was known to be alive 5 years postoperatively). The known number of 5-year survivals is 26 (76 per cent) out of 34 patients followed. The survival time and present condition of the 29 patients known to be living are shown in Table 49.

III. SPINAL CORD ASTROCYTOMAS

There were 11 astrocytomas of the spinal cord. The average age of the patients was 30 years; the eldest was 51 years old and the youngest 8 years. Nine were males and 2 were females.

Three of the lesions were located predominantly in the cervicomedullary and cervical regions, 3 in the cervicothoracic, 4 in the thoracic and 1 in the

TABLE 49

Morbidity in 29 cases of cerebellar astrocytoma—patients now living

Case No.	Present Condition	A.S.E. Code*	Survival to Date
Normal—12 (41%) cases			
3	Normal, works full time	444	13 yrs. 10 mos.
7	Normal, works full time	444	12 yrs. 6 mos.
8	Normal, goes to school	444	12 yrs. 5 mos.
6	Normal, works as a telephone operator	444	12 yrs.
18	Normal, works, has mild hyperthyroidism	444	10 yrs.
41	Normal, works full time	444	7 yrs. 10 mos.
39	Normal, works full time	444	7 yrs. 7 mos.
21	Normal, works full time	444	7 yrs.
24	Normal, goes to school	444	6 yrs.
33	Normal, goes to school	444	5 yrs. 6 mos.
28	Normal, works full time	444	4 yrs. 9 mos.
32	Normal, goes to school	444	4 yrs. 6 mos.
Residual Disability but Self-Supporting—14 (48%)			
9	Slight cerebellar symptoms	344	10 yrs.
1	Weakness of leg and slight ataxia	334	13 yrs. 6 mos.
14	Poor vision in one eye	334	9 yrs. 6 mos.
4	Muscle recession for strabismus	344	13 yrs.
13	Hemiparesis and paresis Vth nerve	334	9 yrs. 6 mos.
37	VII paresis	334	8 yrs. 7 mos.
43	Tires easily	334	7 yrs. 6 mos.
31	Emotional lability	334	4 yrs. 9 mos.
35	Totally blind	234	8 yrs. 6 mos.
12	Hemiparesis	343	10 yrs.
30	Difficulty in walking, holds head to one side	333	4 yrs. 9 mos.
19	Strabismus and headaches	333	9 yrs. 3 mos.
5	Very poor vision, goes to school for blind	232	12 yrs.
23	Very poor vision, does well at school	232	6 yrs.
Residual Disability and not Self-Supporting—3 (11%) cases			
2	Blind in right eye, mild right-sided cerebellar signs, could work but does not	330	14 yrs. 2 mos.
27	Severe cerebellar symptoms	320	6 yrs.
40	Hemiparesis, emotional change, blind	220	8 yrs.

* A=anatomical, S=symptomatic, E=economical: 4 is a perfect score; 0 is complete failure. A perfect score means there is no major organic symptom or sign and the patient can work.

thoracolumbar region. The average duration of symptoms in the 3 cervical and 4 thoracic cases, in which the predominant symptom was one of weakness or clumsiness of one or more limbs, was 6 months. The average duration of symptoms in 3 cervicothoracic and 1 thoracolumbar cases, in which the predominant complaint was pain, except in 1 case, was 2 years and 3 months.

Motor signs took the form of complete paraplegia in 4 patients, and signs of upper motor neurone involvement in 6 others. One patient (No. 2) had a lower motor neurone lesion in the right leg. Nine patients had an area of anaesthesia: in 2 cases only a localized area was involved, as though the tumour was restricted to a narrow region around the point of entry of the posterior roots on one side, while in the other 7 the lesion produced an upper

sensory level either unilaterally or bilaterally, related, presumably, to bilateral impairment of cord transmission. In every case the lesion was thought at surgery to extend slightly further up the cord than the sensory level would indicate, and this is to be expected.

Lumbar punctures were performed in every case, and either a myelogram or a manometric examination and protein studies were carried out. Table 50 shows how the spinal fluid protein value increases as the level of the block descends the spinal cord.

In 4 cases, a large cyst was present. Several small cysts were present in 1 additional case. Microscopic calcium was present in 2 cases. No mitoses were seen in any of the tumours.

Eleven patients were operated upon. Two died in the immediate postoperative period, a case mortality of 18 per cent, and 1 patient was operated upon twice. In both these patients who succumbed (Nos. 3 and 8) the tumours were situated in the cervicomedullary region, and neither was completely removed. A third patient (No. 11), with a cervical tumour, had a biopsy only, and died 1 month after operation, at home. Thus, no patient with a purely cervical lesion lived longer than 1 month after operation.

Three patients with cervicothoracic lesions had a biopsy (No. 1), or a partial removal (Nos. 7 and 9). Two of these patients were given deep roentgen-ray therapy. One of them only, the patient (No. 1) who had a biopsy, is alive and works every day, but is paraplegic (4200 r tumour dose

TABLE 50

Cerebrospinal fluid studies—spinal cord astrocytomas (11 cases)

Region and Case No.	Myelogram	Manometric	CSF Protein Value
Cervicomedullary			
3	—	Almost complete block	—
8 (C1-T2)	—	Complete block	557%
Cervical			
11	—	Complete block	470%
Cervicothoracic			
1	Complete block	Complete block	280%
7	Almost complete block	—	—
9	Almost complete block	—	1160%
Thoracic			
2	Complete block	Partial block	1480% and 1280%
4	Complete block	Partial block	—
6	Complete block	Complete block	—
10	Complete block	Complete block	1664%
Thoracolumbar			
5	—	Complete block	3712% 16% above block

units were given). Another patient (No. 9), who had a partial removal, died 3 years after operation, having received 4700 r tumour dose units. Patient No. 7 had a partial removal, but no roentgen-ray therapy, and died 5 years and 5 months after operation.

Of the 4 patients with thoracic astrocytomas, 2 had apparently complete removal of tumour and roentgen-ray therapy. One (No. 10) is well and almost asymptomatic 7 years after operation and he is working and earning his living (tumour dose, 5500 r). One (No. 4) died 18 months after operation (tumour dose, 4200 r). The third (No. 2) had a biopsy only (tumour dose, 3500 r), but has been lost to follow-up. The fourth (No. 6) had an incomplete removal, no roentgen-ray therapy, and died 6 months after operation.

One patient (No. 5) with a lesion in the thoracolumbar region, had a biopsy and no roentgen-ray therapy. He has been lost to follow-up.

The results of therapy in cases of spinal cord astrocytomas are summarized in Table 51.

It is clear that the outlook in the cervical case is extremely poor, and that surgery has virtually nothing to offer. Almost the only thing that could be done would be drainage of a cyst followed by roentgen-ray therapy. However, roentgen-ray therapy itself causes oedema of the spinal cord tissue, and it is likely that the swelling produced by this treatment alone would be enough to result in medullary paralysis. The outlook for this type of tumour —as for all tumours in that region—is therefore extremely poor.

TABLE 51

Results of therapy—spinal cord astrocytomas*

Region and Case No.	Surgical Therapy	Postoperative Roentgen-ray Therapy	Result
Cervical			
11	Biopsy only	0	Died 1 mo. postoperative
Cervicothoracic			
1	Biopsy only	4200 r	Paraplegic; works 12 yrs. 7 mos. postoperative
7	Partial removal	0	Died 5 yrs. 5 mos. postoperative
9	Partial removal	4700 r	Died 3 yrs. postoperative
Thoracic			
2	Biopsy only	3500 r	Lost to follow-up
4	Complete removal	4200 r	Died 18 mos. postoperative
6	Incomplete removal	—	Died 6 mos. postoperative
10	Complete removal	5500 r	Almost asymptomatic, earns a living. Has diabetes 7 yrs. postoperative
Thoracolumbar			
5	Biopsy	—	Lost to follow-up

* Excluding hospital deaths, Nos. 3 and 8.

In the cervicothoracic region the situation is not quite so hopeless. One patient is still alive and working 12½ years after operation, while 2 other patients (Nos. 7 and 9) died an average of 4 years and 2 months after operation. The patient who had roentgen-ray therapy did not live as long as the patient who had none.

In the thoracic region the situation is similar. One patient is alive after a complete removal of the tumour and roentgen-ray therapy. Two patients lived an average of 1 year after operation—the patient who had roentgen-ray therapy lived longer than the one who did not.

Of 11 patients, therefore, 2 died postoperatively, while the remainder lived 1 month, 6 months, 1½ years, 3 years, 5½ years, 7 years (alive and well) and 12½ years (alive, but paraplegic). Two patients have been lost to follow-up.

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SOME OBSERVATIONS ON THE PROPHYLACTIC USE OF PROCAINE-PENICILLIN

IN OPERATIONS FOR PROTRUDED INTERVERTEBRAL DISK WITH SPINAL FUSION

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As has been noted by Porritt and Cruickshank,[10] and Pulaski and Shaeffer,[11–13] who have reviewed the literature pertaining to antibiotic prophylaxis in surgery, there are relatively few data available on the results of such prophylaxis in surgical cases in which there is no infection. Pennybacker *et al.*,[9] in 1947, demonstrated that the insufflation of penicillin into wounds at the time of closure appeared to reduce the incidence and severity of infections occurring among neurosurgical patients postoperatively. Matera,[6] studying patients who had undergone neurosurgery, and Eriksen and associates,[3] reporting on patients who had undergone thoracic operations, noted a reduction in postoperative infections in wounds when penicillin was used as a prophylactic agent. McKittrick and Wheelock[7] studied the effect of prophylaxis with antibiotic agents in a small group of patients undergoing abdominal operations, and were unable to demonstrate benefit therefrom. Hoerr[4] concluded that penicillin administered prophylactically does not lower the incidence of postoperative pulmonary infections.

Evaluation of the possible benefits of antibiotic prophylaxis in surgical cases in which there is no infection should include consideration of the possible disadvantages of such prophylaxis. Serious allergic reactions to penicillin, including fatal anaphylactic shock, occasionally occur. Needham and one of us (Nichols)[8] found that in hospitals in which large amounts of antibiotic agents are used, a high percentage of the strains of *Micrococcus pyogenes* that are isolated are now found to be resistant to multiple antibiotic agents. Furthermore, Yow[14] noted that the use of antibiotics may suppress the growth of antibiotic-sensitive bacteria in the body, permitting overgrowth of antibiotic-resistant organisms capable of producing lethal infections. Another theoretic disadvantage to prophylaxis with antibiotics, according to Curreri and associates,[1] is that bacterial growth may be merely suppressed and adequate treatment may be delayed because the usual clinical picture

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of infection may be masked and hence not detected at once.[2] Moreover, Long[5] has expressed fear that reliance on antibiotic agents may result in neglect of the fundamental principles of good surgical judgment and technic.

Therefore, an attempt has been made to evaluate the benefits derived from the prophylactic use of penicillin in a type of surgical condition in which infection was absent, but which seemed particularly vulnerable to postoperative infection. Penicillin had been selected as a prophylactic agent because the bacterial spectrum against which it is useful includes those organisms usually responsible for postoperative infections of wounds in the group of patients studied.

MATERIAL

The records of all patients operated upon at the Mayo Clinic in 1953 for protruded lumbar intervertebral disks who also underwent spinal fusion were reviewed. Of these patients, 288 patients had been given prophylactic intramuscular injections of at least 300,000 units of procaine-penicillin each day for several days. For comparison, a group of patients was selected who had undergone the same operative procedure, but prior to the widespread availability of penicillin. In 1943, 119 patients underwent operation for protruded lumbar intervertebral disk and spinal fusion, without prophylactic chemotherapy or antibiotic treatment. Preoperative and postoperative care of the patients otherwise was similar.

METHOD

To evaluate any differences between these two series of patients, the patients were classified according to degree of fever as indicated on the nurses' graphic record (temperatures taken orally, unless specified otherwise), and also according to the condition of the wounds, such as "healed," "imperfect" (that is drainage of any sort from the wound), and "purulent," based on descriptions taken from the notes of the attending surgeons.

RESULTS

The relationship of fever occurring postoperatively to the healing of wounds in these two series of patients is summarized in Table 1. In 1943,

TABLE 1

Relationship of primary healing of wounds to fever among 119 patients who did not receive procaine-penicillin prophylactically (1943) and 288 who did (1953)

Temperature	Patients No.	Wounds Healed No.	Per Cent
No prophylaxis (119 patients), 1943			
Afebrile	35	30	85.7
100°–100.8°F.	32	27	84.4
101°F. or more	52	38	73.2
Prophylaxis (288 patients), 1953			
Afebrile	97	91	93.9
100°–100.8°F.	139	129	92.9
101°F. or more	52	40	76.8

healing *per primam* occurred in approximately the same number of patients in the group with a temperature of 100.0° to 100.8°F. as in afebrile patients. However, among patients with a temperature of 101°F. or more, a percentile reduction in wounds that healed *per primam* is apparent. Moreover, in the same table it is seen that among patients treated in 1953, who did receive procaine-penicillin prophylactically, a similar significant percentile reduction occurred in the healing of wounds *per primam* when the temperature was 101°F. or more. Hence, since a temperature of 101°F. or more appears to have some prognostic significance in the healing of wounds, in our subsequent discussion we have referred to such fever or elevation of temperature as "significant fever."

In Table 2 the over-all result of the study is summarized. Among the 119

TABLE 2

Results when procaine-penicillin prophylaxis was not used (1943) and when it was used (1953)

Complication	Group of No Prophylaxis 1943 (119 Cases) Per Cent	Group of Prophylaxis 1953 (288 Cases) Per Cent
Purulent drainage	5.9	1.7
Significant fever	43.7	18.1
Imperfect wounds*	20.2	9.7

* Includes purulent and serous drainage.

patients in 1943 who did not receive antibiotic prophylaxis, purulent drainage from the operative site developed in 7 or 5.9 per cent. Among the 288 patients who received penicillin prophylactically in 1953, purulent drainage subsequently developed in 5 or 1.7 per cent. In 1953, as compared with 1943, there was a reduction in significant postoperative fever by about a half, and there was a similar reduction in the number of patients with imperfect wounds (Table 2). In the 1953 group there was no patient whose life was endangered by infection; however, in 1943 septicemia caused by *Micrococcus pyogenes* developed in one patient, complicating his infected wound, and his life undoubtedly was saved by the intravenous administration of penicillin. This patient was the only one in the 1943 group to receive penicillin during his hospital stay. There were no deaths in either group of these cases.

There were 15 probable untoward reactions to penicillin in the 1953 group, providing an incidence of 5.2 per cent. These reactions were limited to pruritic rashes, and none of them was considered to be of an immediately serious nature. Inasmuch as many of the patients were receiving other drugs as well as penicillin, the exact incidence of reactions to penicillin could not be determined.

Some incidental observations may be of interest. In 1943, 5.9 per cent

of the patients had thrombotic or embolic complications, whereas in 1953 only 2.4 per cent had such complications. In 1953, 12 patients received cortisone preoperatively and postoperatively because of possible adrenal insufficiency arising from the previous administration of cortisone, and in all of these cases primary healing of wounds occurred. In the 2 years of the study, imperfect wounds and instances of fever were evenly distributed throughout the 12 months of each year.

COMMENT

The percentile occurrence of purulent infections, significant postoperative fevers and imperfect healing of wounds was significantly less among the patients who underwent surgical operations in 1953 as compared with those who had undergone operations in 1943. With the exception of the prophylactic administration of penicillin to members of the 1953 group, preoperative and postoperative care of the patients apparently was similar in each series.

It is recognized that sweeping conclusions can not be drawn on the basis of these comparisons, inasmuch as some uncontrollable variables do enter into such a study. Moreover, as noted previously, the nature of the particular surgical procedures studied renders patients more than usually vulnerable to postoperative complications of wounds. There is always some bleeding about a bone graft, and the graft itself is essentially a foreign body, both of which factors may predispose these patients to infections.

It should be emphasized that any allergic reaction to penicillin may interdict its future use entirely. Employment of an antibiotic agent prophylactically, an action that might vitiate the use of that agent in the future for potentially lethal infections, actually may be false medical economy.

We believe the decision as to whether the possible benefits of the prophylactic use of penicillin outweigh the possible disadvantages of the agent should be made in each case.

The close correlation between the incidence of significant fever (that is, a temperature of 101°F. or more) in the postoperative state and the occurrence of imperfect healing of the wound also is of interest. It would appear that significant fever in the postoperative state is an indication for more intensive antibiotic therapy at that time, if causes of fever, other than possible infection of the wound, have been excluded.

Continuation of the discriminate use of antibiotics as prophylactic agents against infection in this type of surgical procedure seems warranted as an investigative procedure.

SUMMARY AND CONCLUSIONS

The records of 288 patients who underwent surgical operations in 1953 at the Mayo Clinic for protruded lumbar intervertebral disk and spinal fusion, and who had received procaine-penicillin prophylactically, were reviewed. For comparison, the records of 119 patients who had undergone the same operative procedure in 1943, but without chemotherapeutic or anti-

biotic prophylaxis, were studied. It was found that the incidence of purulent infection of operative wounds was reduced from 5.9 per cent in 1943 to 1.7 per cent in 1953. In cases in which the temperature was recorded as 101°F. or more, there was a significant reduction in the frequency of primary healing of wounds. In 1953, when antibiotic agents were used, as compared with 1943, when they were not used, there was a reduction in significant postoperative fever by about a half, and there was a parallel reduction in the frequency of occurrence of imperfect wounds. While they are not conclusive, we believe these observations may be of value when the benefits of antibiotic prophylaxis in surgical cases devoid of infection are being considered.

The possibility of untoward reactions to penicillin is sufficiently great that, in evaluating the feasibility of employing antibiotic prophylaxis, the physician should not overlook this real disadvantage.

Additional studies which may help to clarify the problem of antibiotic prophylaxis in this type of surgical procedure appear indicated.

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THE VALUE OF THE ELECTROENCEPHALOGRAM IN SELECTED CASES OF SUBDURAL HEMATOMA

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In a previous communication[1] it was shown that the initial electroencephalogram (EEG) was of no value in differentiating the operative from the nonoperative type of head injury, insofar as unilateral subdural hematomas in adults were concerned. The parameters of reduction in amplitude with or without a slow wave focus in the EEG were present in these two groups of head injuries regardless of whether the initial EEG was taken in the acute, subacute, or chronic stages.

It would therefore seem that the EEG would have only limited value from the standpoint of identification of unilateral subdural hematomas in adults. However, as a corollary its use is much more important from a negative standpoint; that is, given an individual who has had a head injury and who does not present the criteria of reduction in amplitude with or without a slow wave focus, the possibilities are good that there is no subdural hematoma present.

There is an interesting exception to the above, in those instances in which a subdural hematoma is present on the same side as the focal neurologic symptoms and signs. In the previously reported group of 60 cases there were 6 patients who had a subdural hematoma with paresis on the same side as the lesion (here called ipsilateral subdural hematomas). Since that communication we have had an additional 6 cases. In all cases the hematomas and the focal signs were present on the side of the amplitude reduction and delta focus. In brief, it may be stated that given an individual, who has had a head injury and who manifests focal neurological signs such as a hemiparesis, if there is an amplitude reduction in the EEG on the same side the possibilities are good that a subdural hematoma is present on that side. Although this finding may be of limited value in large accident rooms where comatose patients frequently arrive with no history or evidence of trauma, the presence of focal neurologic signs coupled with an ipsilateral reduction of amplitude on the EEG, should make one strongly suspicious about the possibility of subdural hematoma on the side of the decreased voltage.

It is the purpose of this communication to report on the positive value

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of the EEG in the identification of unilateral subdural hematomas in adults. Further, it will be demonstrated that some EEG epochs better exemplify amplitude asymmetry than others.

PRESENT STUDY

In the present studies the EEG findings in 12 proven cases of ipsilateral subdural hematoma were reviewed. Serial EEGs were examined for additional diagnostic clues.

The EEGs were performed on a Grass 8-channel ink-writing oscillograph with a minimum of 13 needle (27 gauge) electrodes inserted into the scalp and ear lobes using the following references:

(1) Paired symmetrical electrodes referred to both ears.

(2) All electrodes on the left side referred to the left ear and those on the right to the right ear.

(3) Similar to (2) above except that the reference point was a common vertex placed in the midsagittal plane halfway between the frontal and parietal electrodes.

(4) Bipolar triangulation (here called short intercepts).

RESULTS AND DISCUSSION

Figs. 1–3 demonstrate 2 cases in which ipsilateral hematomas were evacuated subsequent to electroencephalography. In each case there was reduc-

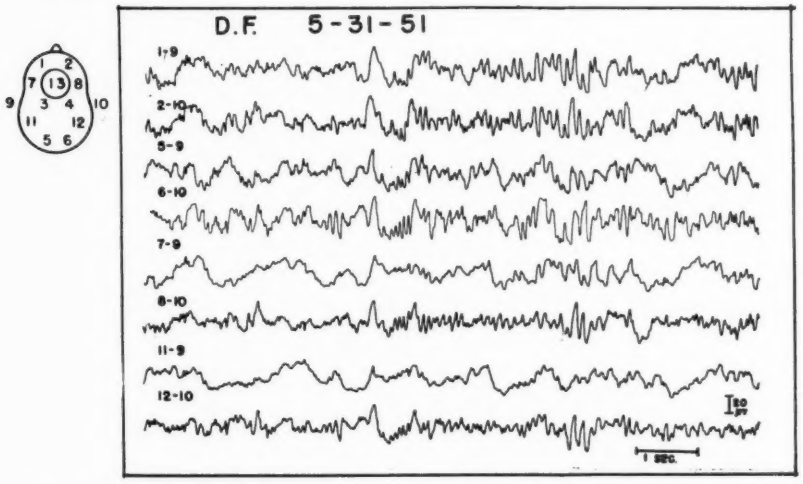

FIG. 1. D.F. was found comatose on May 12, 1951. She had left 3rd nerve paralysis, left hemiplegia and bloody CSF. There was initial clinical improvement. EEG shows reduction in amplitude and a delta focus on the left side, maximal in the temporal region. Note also the depression of the sleep spindles on the left side. Because of these findings, and in spite of the left hemiplegia, a left carotid arteriogram was done and demonstrated a picture compatible with a subdural hematoma on this side. The patient remained comatose subsequent to arteriography and a subdural hematoma was evacuated on the left side. She made a complete recovery.

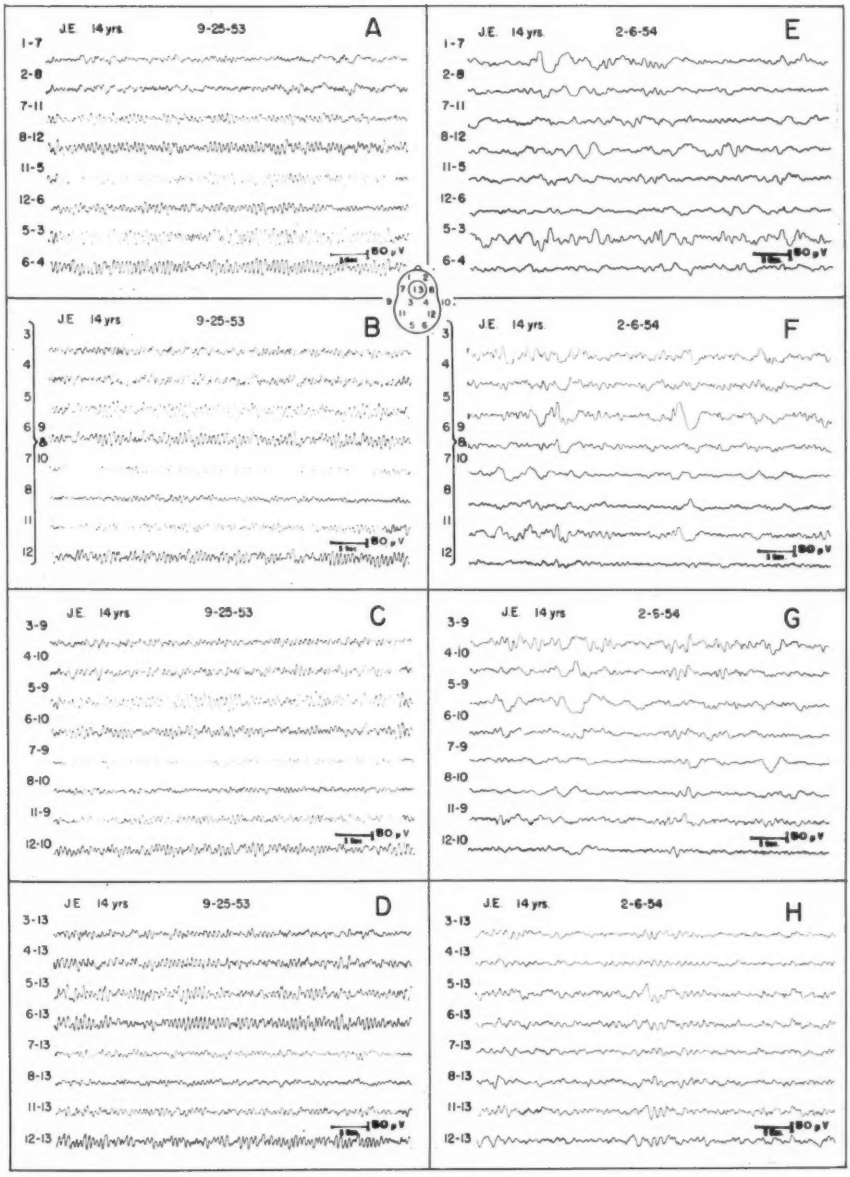

FIG. 2. This demonstrates the importance of taking the various epochs in the EEG. Of particular importance, it shows that the amplitude asymmetries are invariably best marked in short intercepts (homologous triangulation). See Fig. 3 for history. (A-D) Various epochs taken on the same individual prior to any known head injury and offered as a control. Note that there is no remarkable amplitude asymmetry and particularly not in the short intercepts episode. (E-H) A similar series on the same individual, taken 5 days post trauma and 6 days preoperatively. Note that there is a generalized slowing in the EEG and that the decrease in amplitude and minimal slow wave focus is most marked on the right side in the short intercepts episode (Fig. 2E).

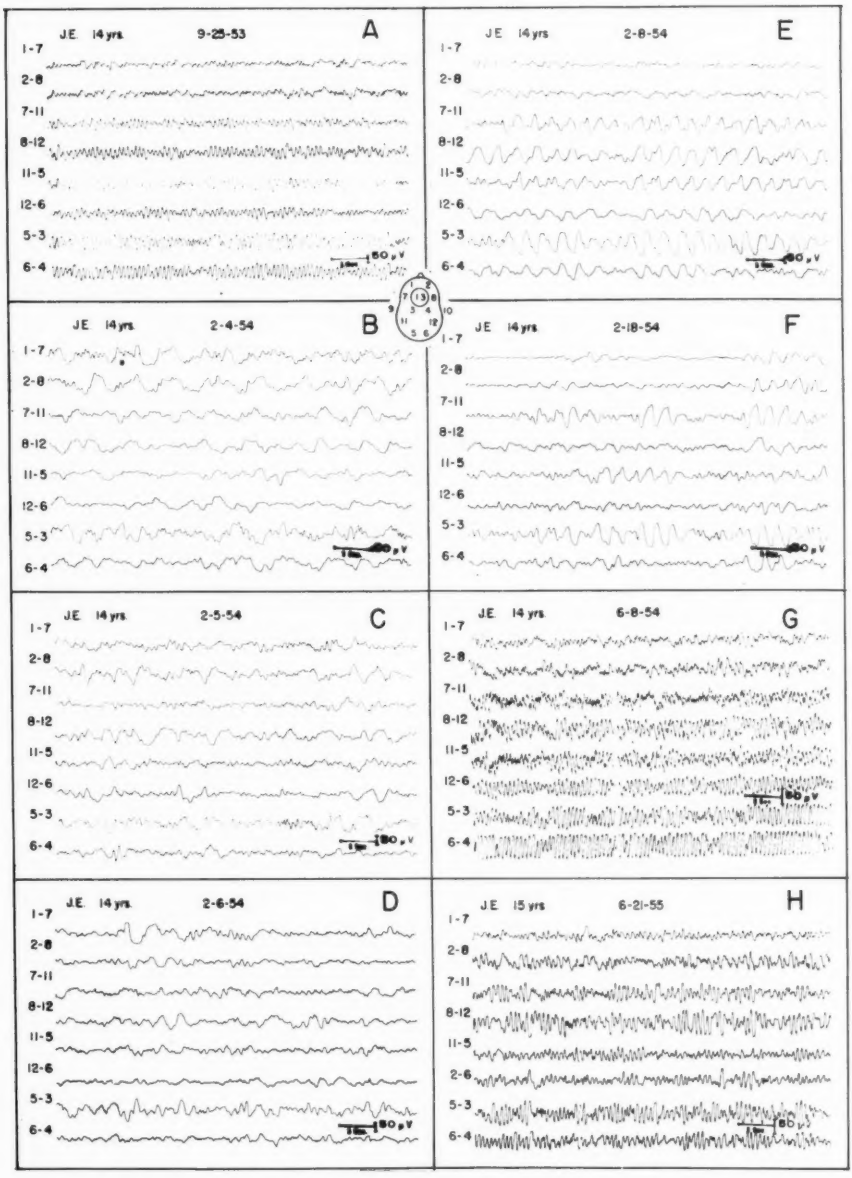

FIG. 3. J.E., 14-year-old white male, had osteogenesis imperfecta with a history of numerous fractures to various long bones of the body. He had had syncopal episodes since August 1953. On Feb. 1, 1954 he suffered a head injury, followed by development of a right hemiparesis and decreasing mental awareness. On Feb. 12, 1954 bilateral burr hole exploration was made and a large subdural hematoma

tion in amplitude and a mild to moderate slow wave focus on the side of the hematoma.

In our experience the epoch that best shows the amplitude asymmetry (unilateral reduction in voltage) is the one that we call short intercepts (Fig. 2A). The episode that seems to show this next best is the one in which all electrodes on one side are referred to the ipsilateral ear and compared to the opposite side. The epoch of least value from the standpoint of amplitude asymmetry is that in which the vertex is used as a common reference point. However, this latter epoch shows seizure activity better than any of the other episodes.

As regards amplitude asymmetry it should be noted that reference is being made only to the reduction of background activity. Occasionally medium voltage slow waves will be seen which when measured from trough to crest actually exceed the voltage on the opposite side. However, the faster background activity is always reduced when compared to the opposite side. Amplitude reductions are usually best seen in the temporal and occipital areas. Sleep spindles are definitely reduced on the side of the subdural hematoma (Fig. 1).

Seizure activity, when present, may be confusing in cases of acute subdural hematomas. It is usually most prominent on the opposite side (Fig. 1).

Serial EEGs may be of value in the diagnosis of ipsilateral subdural hematomas, in instances in which the patient, after an initial period of coma, may improve for hours or even days. Occasionally this improvement may be paralleled by improvement in the EEG. However, the persistence of an ipsilateral reduction in amplitude with or without a slow wave focus would still point very strongly to the possibility of a subdural hematoma (Fig. 3B, C, D).

The voltage reduction follows a pattern similarly described in other conditions such as ruptured aneurysms and thromboses.[2,3] The initial effect following injury is a reduction in amplitude on the side of the lesion. Once the irritating substance is removed several courses may be followed. In the

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was evacuated from the right side. No abnormalities were noted on the left. Subsequently he completely recovered. Note the following:

1. 3A, taken on Sept. 25, 1953, appears as pretraumatic control and is considered to be a normal EEG.

2. 3B, taken 3 days post trauma, shows a decrease in amplitude on the right side, generalized slowing of the EEG, and a delta wave focus on the right.

3. There was subsequent improvement in both the clinical status and the EEG, although the decrease in amplitude and slow wave focus on the right side persist (3C and 3D).

4. Worsening of the patient's condition began on Feb. 8, 1954 and continued until the time of operation on Feb. 12, 1954 when he became comatose. The increase in generalized slow activity is noted but the persistent decrease in amplitude on the right side is emphasized (3E).

5. There was subsequent improvement in the EEG during the next 16 months. 3F shows that the record is still slow 4 days postoperatively and the amplitude on the right side is quite markedly reduced. 3G, taken 4 months later, now shows the amplitude is increased on the right side, particularly in the temporal-occipital electrodes. 3H, taken 16 months postoperatively, shows the permanent reduction in amplitude on the right side.

first place, the amplitude may remain reduced indefinitely. Secondly, the initial reduction of amplitude, which may persist for only 10 to 14 days, is replaced by an ipsilateral increase in amplitude for a similar period of time and then reverts to reduction. Thirdly, the EEG may go through several amplitude fluctuations before a final permanent reduction (Fig. 3E, F, G, H).

SUMMARY

Twelve cases of unilateral subdural hematomas in adults are presented in which there was the unique finding of ipsilateral hemiparesis, that is, hemiparesis on the side on which the subdural hematoma was located. The electroencephalographic picture showed a reduction in amplitude and a delta wave focus on the side on which the subdural hematoma was located.

It is stressed that, given the criteria of an individual in coma with or without the history of a head injury, the presence of reduction of amplitude and slow wave focus on the same side as the focal neurologic signs, suspicion should be strong for the possibility of an extracerebral lesion such as a subdural hematoma.

Evidence is given that the amplitude asymmetry shows up in some epochs better than in others and the best combination is one in which symmetrical areas are triangulated.

In spite of an improving EEG, from the standpoint of disappearance of generalized delta activity, persistence of ipsilateral reduction in amplitude and slow wave focus in an individual who has had a head injury, points strongly to the possibility of a subdural hematoma on the side of the amplitude reduction.

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PARATRIGEMINAL EPIDERMOID TUMORS

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PARATRIGEMINAL epidermoid tumors are uncommon but their recognition is important because prompt and proper treatment is associated with excellent results in contrast with many of the other tumors found in this location. We have encountered 2 such tumors. As a survey of the literature has revealed only 1 other, reported by King,[13] a summary of his case is also included. In our first case the lesion was not correctly diagnosed pre-operatively. However, in the second case it was recognized clinically because of its resemblance to the first. In Dr. King's case a diagnosis of probable meningioma of the middle fossa was made by him and by Dr. Foster Kennedy. It will be seen that with the signs and symptoms plus the radiographic changes a correct pathologic diagnosis may be suspected pre-operatively.

This article deals mainly with epidermoids—so-called cholesteatomas or pearly tumors. To our knowledge the closely related dermoids containing other elements of skin such as sweat glands, sebaceous glands, and hair follicles, have not been found in the area with which we are concerned here. Intracranial epidermoids are relatively rare neoplasms and constitute, according to various figures, between 0.2–1.0 per cent of all intracranial tumors. Mahoney[17] stated that the first intracranial tumor of this type was depicted by Pinson[20] who in 1807 prepared a wax model of such a tumor in the cerebellum and fourth ventricle. Bailey[1] in 1920 collected some 62 cases of which many were doubtful, and Grant and Austin[10] in 1950 reported 22 seen since 1930.

Sex incidence is difficult to determine as in many papers such details are not given, but in those in which the sex of the patients was disclosed, there were 35 males and 26 females. The range in age was from 11–71 years and the average age at the time of surgery as calculated from 39 cases was 35.6 years. The average duration of symptoms was 16 years. In Grant's[10] series the average duration of symptoms was 2.9 years in the extradural group, and 4.6 years in the intradural epidermoid group.

These are very slowly growing, space-occupying lesions generally agreed to be congenital in origin.[21] They may be epicranial, intradiploic, epidural, intraventricular or intracerebral. The consensus is that they are ectodermal

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rests, remnants of incomplete separation of neural from surface ectoderm. These fetal inclusions of epidermal cells, depending upon the depth of the layer and/or the embryonic age, result in either dermoids or epidermoids. Closure of the medullary groove in the human embryo occurs at the third week when constrictions appear resulting in the formation of primary cerebral vesicles. During the fourth and fifth week secondary vesicles are developing and through them cell rests may be misplaced away from the midline. The process of formation of the optic and otic vesicles may contribute substantially to the resultant congenital epithelial tumors found in more peripheral locations. It seems likely that the paratrigeminal epidermoids to be discussed here had their origin from embryonic rests associated with the primary otic vesicles.

Intradiploic epidermoids, according to Bucy,[3] are usually found in the frontal and parietal regions. Intracranially epidermoid tumors are found most frequently in the extracerebral spaces along the base of the brain and in the cerebellopontine angle, although Bailey[1] presented 8 cases in which epidermoids were found in the fourth ventricle and 1 in the third ventricle. The symptoms they present are those of any slowly growing tumor. Or they may be found unexpectedly at autopsy. The radiographic appearance is so typical that most intradiploic epidermoids are correctly diagnosed preoperatively. Intracranial extradural tumors may, likewise, present a typical picture of sharply defined bony destruction. In Grant and Austin's[10] series of 7 extradural epidermoids all showed typical radiographic changes, but of 15 intradural tumors the roentgenograms were of no help in the recognition of 14.

The inner lining of the tumors usually presents a smooth, silky, irregular grey or mother-of-pearl surface. The contents are generally solid, cheesy, dry, and grumous, but occasionally may be a thick, viscid, dark brown or grey material containing cholesterol crystals. In the diploe the epidermoid may have a reddish-pink appearance which King[13] compared to the lining of boiled lobster muscle. The tumors that most closely resemble them are dermoids, as previously mentioned, and the adamantinomas which, however, contain teeth anlage and may contain embryonic teeth that are visible radiologically. Other conditions to be differentiated from the neoplastic epidermoids are the chronic infectious cholesteatomas of the middle ear to which these have no relation but with which they are often confused. Radiologically the intradiploic lesions may also be confused with sarcomas of the skull, eosinophilic granulomas, and osteitis fibrosa cystica.

Microscopic examination of these tumors reveals a flat stratified squamous epithelium resting on a supporting connective-tissue membrane without papillae. There are no dermal elements, such as sweat and sebaceous glands and hair follicles. The surface of the epithelium is turned inward so that as the surface cells are continuously cast off they form the cheesy interior of the tumor which gradually increases in size.

Theoretically the ideal treatment of any epidermoid is complete removal.

However, with intracranial epidermoids part of the capsule is often so adherent to important structures that a complete removal is not feasible. Fortunately recurrence is rare even if part of the capsule is left behind.[7,18] In Grant and Austin's[10] series of 22 epidermoids 8 were completely removed. These included 4 of 15 that were intradural, and 4 of 7 extradural. With the 7 extradural tumors there was no mortality. However, the over-all mortality rate in Grant's series was 22.8 per cent, which agrees closely with Love and Kernohan's[16] mortality of 21.4 per cent with dermoids and epidermoids of the central nervous system. Grant makes the point that the operative mortality is higher than with meningiomas because the epidermoids tend to lie closer to important midline structures, they grow more slowly and fail to produce symptoms until a late stage, and because they seldom occupy the favorable position at the cerebral convexity so characteristic of the meningiomas. Postoperatively the main complication, aside from what might be encountered with any intracranial tumor, is a sterile meningitis caused by irritation of the leptomeninx by the contents of the epidermoid. This may be avoided by thoroughly removing all such material and irrigating the area with saline.

In the following 3 cases of paratrigeminal epidermoid tumor the petrous pyramid was eroded in each instance, and it is thought that the tumor arose there and burst out into the middle fossa beneath the gasserian ganglion. In another case, to be reported elsewhere, the epidermoid tumor appears to have also arisen in the petrous pyramid producing an erosion similar to that seen here but extended backward into the posterior fossa just medial to the internal auditory meatus.

CASE REPORTS

Case 1. No. 4606. W.K., a 36-year-old white male university professor, was referred by Dr. M. M. Hipskind of Chicago. He was admitted to the University of Chicago Clinics on Oct. 24, 1939 complaining of decreased vision in the right eye and numbness of the right side of his face.

He had been seriously injured in an automobile accident in which he sustained a basal skull fracture 11 years previously. Soon after this accident he noticed paralysis of the jaw muscles on the right side and some loss of feeling on the right side of the face. At first this was attributed to the skull fracture. These symptoms progressed for 2 years following the automobile accident and then became stationary. Two years before admission he was accidentally struck in the right eye by a twig but noticed no pain, in spite of denudation of the cornea. This was the first time that he realized that the eye was anesthetic. A few months prior to admission he began to notice impaired vision in the right eye which progressed to an inability to recognize people at close range. Recently at irregular intervals he had had sudden but not severe twinges of pain in the right side of the face.

Examination. There was complete paralysis with atrophy of the right temporal and masseter muscles. The jaw deviated to the right when opened. There was anesthesia over the distribution of the right trigeminal nerve and the right corneal reflex was absent. Visual fields showed a small central scotoma in the right eye. Visual acuity corrected was 0.5 minus 2 on the right, and 1.5 plus 2 on the left. Examina-

tion of ear, nose and pharynx disclosed no abnormalities. Blood and urine were normal.

Roentgenograms of the skull (Figs. 1 and 2) revealed a bone-destroying lesion of the floor of the right middle fossa with involvement of the petrous pyramid and the sphenoid. The foramina spinosum and ovale were obliterated. The area of destruc-

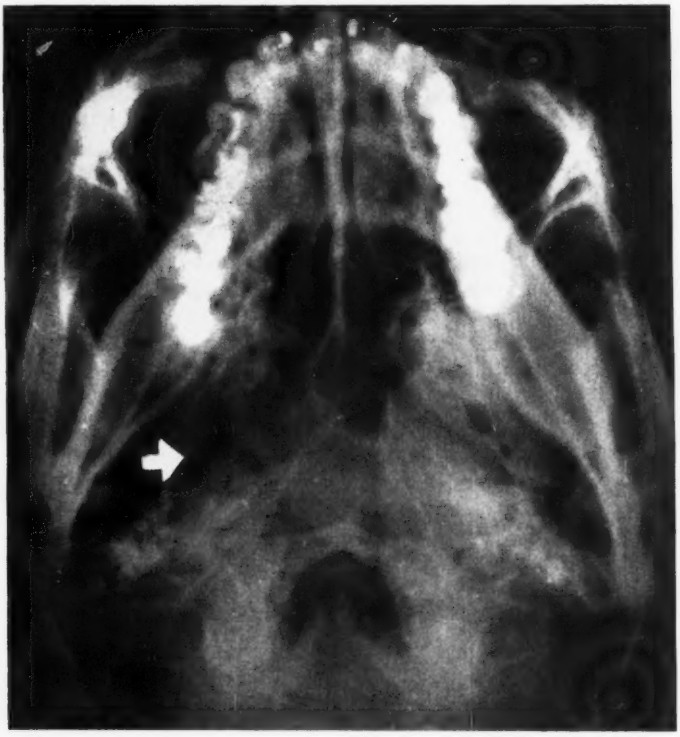

FIG. 1. *Case 1.* Roentgenogram of base of skull. There is marked decalcification of the medial part of the floor of the right temporal fossa and of the anterior surface of the apex of the right petrous pyramid. Arrow indicates margin of decalcification. The foramina ovale and spinosum have been completely obliterated on the right side.

tion of the floor of the skull measured approximately 2.5 cm. by 3.5 cm. in diameter. The apex of the petrous pyramid had been eroded.

1st Operation. On Nov. 21, 1939 a right temporal osteoplastic flap was turned with its base at the zygoma. The dura mater was tense but after ventricular puncture good decompression was obtained. The gasserian ganglion was then approached. The mandibular division was swollen and purple. An extradural epidermoid was found causing a 1-cm. area of destruction of the petrous bone. The tumor extended anteriorly to the region of the optic foramen but neither the carotid artery, the cavernous sinus, nor any of the nerves in this region were exposed. The tumor was removed with a copper spoon, pituitary rongeur, and suction; the capsule was removed easily

from the base but was adherent to the dura mater and this part was not removed. Near the apex of the petrous pyramid the cheesy contents of the tumor had ruptured through the capsule and the dura mater and lay beneath the pons. This part, too, was all thoroughly removed. The cavity was then washed thoroughly with normal saline and the wound was closed after insertion of a drain.

FIG. 2. *Case 1.* Postero-anterior roentgenogram of skull, postoperative. The petrous pyramids have been projected through the orbits and the erosion and decalcification of the apex of the right petrous pyramid are evident. Arrow indicates margin of erosion.

Postoperative Course. The patient did well except for a short period of fever, signs of meningeal irritation, and a partial paralysis of the right facial nerve which cleared rapidly.

Pathological Report. Sixty cc. of the tumor mass were recovered, plus a considerable quantity which was taken up by suction. The cyst wall consisted of stratified squamous epithelium resting on a layer of connective tissue while the bulk of the tumor was made up of the desquamated cellular debris which filled the cyst (Fig. 3).

Subsequent Course. In the summer of 1940 he noticed a bilateral photophobia. In the winter of 1940–1941 progressive visual difficulty began to develop. In De-

cember 1941 Dr. Peter Kronfeld found that the vision was 20/70 in the left eye and 10/200 in the right eye. There was a small central scotoma in the left visual field and a moderate constriction of the right visual field. The right visual field had a moderate scotoma and three other absolute and relative scotomata in the superior temporal quadrant. The optic discs and maculae were normal.

Neurological examination revealed a complete lesion of the right trigeminal nerve involving both sensory and motor components. Roentgenograms showed no further bony erosion of the skull. However, it was obvious that there was some progressive

FIG. 3. *Case 1.* Photomicrograph of wall of epidermoid cyst. The layer of stratified squamous epithelium is shown with the germinal layer below and to the left. There is a mass of the desquamated epithelium that filled the entire cyst. Hematoxylin and eosin, ×100.

intracranial process which was now involving the left optic nerve as well as the right. Its nature could only be surmised, but we naturally thought it most likely that the epidermoid tumor had extended across the midline and was now pressing upon both optic nerves. He was admitted to The Chicago Memorial Hospital.

2nd Operation. On Jan. 9, 1942, a left frontal osteoplastic flap was reflected and the optic nerves and chiasm were explored. There was no evidence of the epidermoid or any other tumor, but the optic nerves and chiasm were incased in a dense, thick layer of connective tissue. It was possible to incise this thickened arachnoid membrane and to peel it off the optic nerves and chiasm.

Postoperative Course. He did well. His visual acuity promptly improved. On June 24, 1942 Dr. Kronfeld found visual acuity in the left eye to be 20/40. On Aug. 6, 1943 the vision in the right eye was 20/800, in the left eye 20/33.

Dr. Harold G. Scheie of Philadelphia reports that since 1947 the patient's vision has been unchanged. When last examined on Oct. 21, 1953 he was able to count fingers with his right eye and the visual acuity in the left eye was 6/22. The visual field of the left eye was full, that of the right showed only a sector of vision remaining in

the lower temporal quadrant. The right optic disc was normal; the left was slightly paler. Ever since the operation the patient has been in good health and has led an active, productive life in teaching and research.

Comment. This development of a dense chiasmatic arachnoiditis is certainly an unusual situation, and the only one of its kind with which we are familiar. It seems practically certain that the irritation of the leptomeninx by the contents of this epidermoid tumor, which were found intradurally beneath the brain stem and which provoked the postoperative sterile meningitis, also induced this hyperplastic process in the pia-arachnoid membrane leading to the intense constricting arachnoiditis around the optic nerves and chiasm.

This case emphasizes the chronicity of these tumors and the good prognosis associated with their surgical removal. These tumors are present from birth yet their growth is so slow that 30 or more years are commonly required for them to reach a size where they produce clinical manifestations that call them to their victims' attention. It is not surprising, therefore, that a complete removal of the cheesy contents of the cyst, even though all of the viable wall is not removed, results in such a favorable outlook. Most of these tumors, so treated, never give rise to symptoms again. In this particular case the operation was performed over 16 years ago and the patient continues in excellent health.

Case 2. A.A., a 38-year-old white female kitchen helper, was admitted on Oct. 5, 1954 to the Chicago Wesley Memorial Hospital when diabetes mellitus and masses in the breasts were found on an employment physical examination. The patient was of low intelligence and had no complaints nor did she think it strange that the left side of her face was found to be anesthetic. On inquiry she was found to have a nocturia of two to three times per night.

Examination. Aside from the low intelligence, positive neurological findings were an absent left corneal reflex, analgesia and thermanesthesia of the left side of the face, paralysis of the motor division of the left trigeminal nerve with atrophy of the left masseter and left temporal muscles, and deviation of the lower jaw to the left on opening the mouth. There was partial paralysis of the left 6th cranial nerve and there was also a slight flattening of the right nasolabial fold. The patient had nystagmus, coarser on lateral gaze to the right and finer on looking toward the left. The right knee jerk was more active than the left. There were decreased associated movements of the right arm when she walked. Babinski's sign was definite on the right, equivocal on the left, and Hoffmann's sign was present bilaterally.

An admission blood count was normal and Kahn test on the blood was negative. The urine was loaded with pus cells and showed a trace of albumin and sugar. Fasting blood sugar was 216 mg. per 100 cc. Serum nonprotein nitrogen was 33.5 mg. per 100 cc. Urea clearance was 33 per cent and sedimentation rate was 36 mm. per hour. An electrocardiogram was normal. Roentgenograms of the skull revealed an erosion of the petrous apex 1.5 cm. wide (Figs. 4 and 5). Intravenous pyelography showed slight dilatation of the calices, pelves and ureters bilaterally.

Course. After the diabetes had been brought under control and the cystitis had been relieved by antibiotics, the tumors of the breasts were removed on Oct. 17, 1954 and found to be benign adenomas.

Operation. On Oct. 18, 1954, under general anesthesia, a left osteoplastic craniotomy was performed. An intradural subtemporal approach was made to the petrous apex. However, it was readily seen that the mass lay extradurally. The tumor was exposed by elevating the dura mater from the floor of the temporal fossa. The tumor capsule was opened and the contents were found to be dry, cheesy, grey material. This was removed without difficulty. Approximately 30 cc. of the tumor contents

FIG. 4. *Case 2.* Roentgenogram, Towne position, showing the striking erosion of the apex of the left petrous pyramid, the margin of which is indicated by the arrow. The erosion includes the margin of the internal auditory meatus.

were saved and considerable was lost down the sucker. The capsule was quite adherent to the dura mater and the floor of the middle fossa. Therefore, only the lateral wall was removed to allow adequate evacuation of the contents. Following evacuation of the tumor the base of the skull was clearly visible, but the only structures that could be definitely identified were the 7th and 8th cranial nerves which passed inferior and posterior to the tumor. The tumor itself appeared to extend from the mid-region of the pons to the anterior end of the temporal fossa. After complete evacuation of the cyst the wound was closed in anatomical layers.

Microscopic examination confirmed our impression that this was an epidermoid cyst (Fig. 6).

Course. Immediately after operation the patient did well but 4 days later she failed to respond adequately and showed definite evidence of a right hemiplegia. It was felt that there was either a subdural or extradural hematoma and the wound was re-opened. There was no evidence of a hematoma and the symptoms were thought to be caused by edema and hyperglycemia. Her postoperative course was stormy. The fasting blood sugar level was very difficult to control and at one time

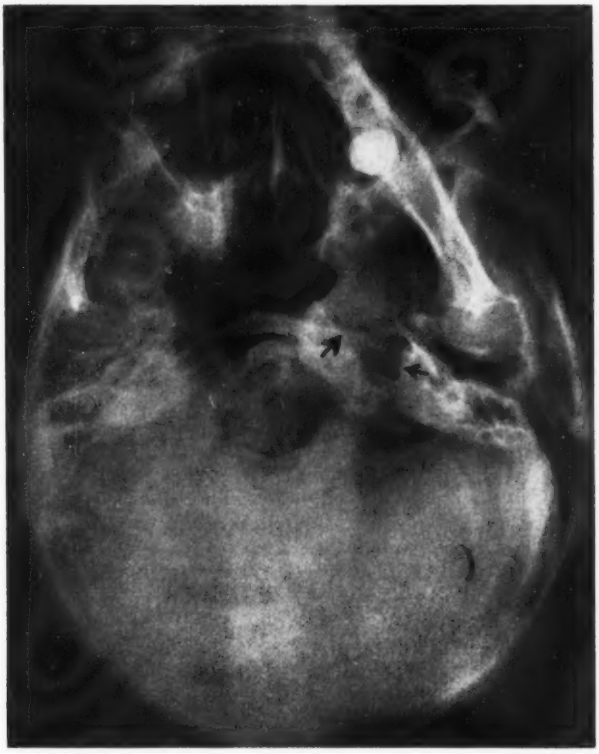

FIG. 5. *Case 2.* Roentgenogram of base of skull, showing erosion of the apex of the left petrous pyramid and of the floor of the left temporal fossa. Arrows indicate margins of the erosion. The foramina spinosum and ovale have been completely obliterated on this side.

reached 426 mg. per cent. The urinary output in the first few days after surgery was between 3000 to 4000 cc. daily. The right hemiparesis and coma gradually cleared. The patient was discharged on Nov. 13, 1954 with a slight residual right hemiparesis, most marked in the face, and a fasting blood sugar of 120 to 150 mg. per cent.

When seen 5 months later she appeared to be more intelligent than pre-operatively. In addition to the anesthesia of the left side of the face, she complained of some loss of memory. There were hypesthesia and hypalgesia of all divisions of the left trigeminal nerve. The left corneal reflex was severely decreased. The left mas-

seter and temporal muscles were atrophic and the jaw deviated to the left when it was opened. There was some facial asymmetry with flattening of the right nasolabial fold and a widening of the right palpebral fissure but there was no weakness of the face on voluntary movements.

Comment. Here again there was a congenital tumor which did not produce symptoms that demanded treatment until the patient was in her fourth

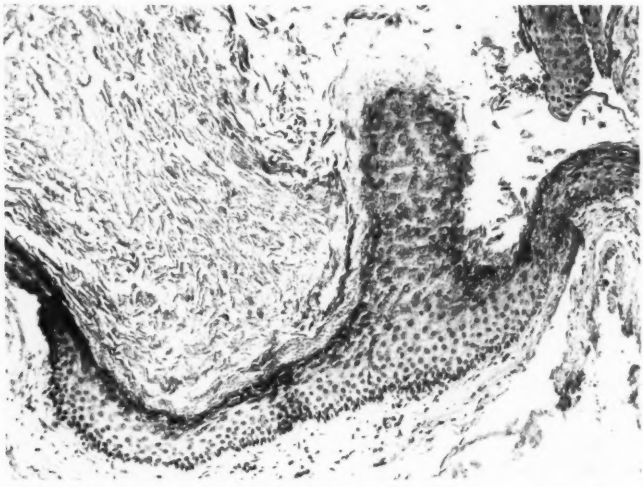

FIG. 6. *Case 2.* Photomicrograph of wall of epidermoid cyst. The layer of stratified squamous epithelium is shown with the germinal layer on its lower margin. Above it lies a mass of desquamated epithelium of the type that filled the cystic cavity. Hematoxylin and eosin, ×100.

decade of life (38 years old). Judging from other cases it is unlikely that the tumor will ever again cause her trouble even though much of its wall remains.

Case 3. A 40-year-old white female was a patient of Dr. Joseph King[13] in 1938. She complained at that time of diplopia of 12 years' duration. Coincident with the onset of this the patient had numbness of the left face and forehead following removal of some impacted teeth on that side. She also had occasional headache in the occipital region and pain in the left eye and the left side of the face. For 9 years she had continuous diplopia with diminution of vision in the left eye. In 1934 she had bilateral myringotomy with profuse discharge. This was followed by bilateral mastoidectomy with relief of her diplopia for 6 months. Prior to admission she had multiple left corneal ulcerations.

Examination. Her visual acuity was 20/15 in the right eye and 20/50 in the left eye corrected. There was a marked contraction of the left central field. There was paralysis of the left lateral rectus muscle and superior-oblique with marked limitation of all other movements of the left eye. The left pupil was fixed and dilated. The left side of the face and the cornea were anesthetic and there was atrophy of the temporal and masseter muscles. She had a partial peripheral type facial palsy on the left and almost complete deafness in the left ear.

Roentgenograms showed severe destruction of the left petrous apex, depression of the floor of the middle fossa, and erosion of the left clinoid processes, sphenoid body, and lower portion of the temporal squama.

Operation. A large epidermoid cyst was encountered and material amounting to approximately 60 cc. was removed. The cyst was estimated to be about the size and shape of a duck's egg. A large part of the capsule was removed, but no attempt was made at total extirpation of the capsule. The carotid artery was exposed and the position of the gasserian ganglion was seen but no cranial nerves could be definitely identified.

Pathologic examination confirmed that this was an epidermoid tumor.

Course. Following an uneventful recovery the patient was discharged mentally clear 15 days postoperatively. The left pupil was slightly irregular. There was a complete left abducens palsy. The left corneal reflex was absent, and there was incomplete paralysis of the motor division of the left trigeminal nerve. There was also a partial left facial paralysis and almost total deafness on that side.

Comment. Information as to this patient's course after leaving the hospital is not available. The similarity of this and our 2 cases is obvious. In all the condition was characterized by a very slow insidious development with primary involvement of the sensory and motor divisions of the trigeminal nerve and evidence in the roentgenograms of erosion of the inner portion of the petrous pyramid and of the floor of the middle fossa, particularly in the region of the foramen ovale. In addition, other cranial nerves and adjoining bony structures were involved. Signs of generalized increased intracranial pressure were not present in these 3 cases.

DISCUSSION

Trigeminal and paratrigeminal tumors themselves are not extremely common and may be of many varieties including neuroma, meningioma, neurofibrosarcoma, sarcoma, metastatic carcinoma, chondroma, aneurysm, and epidermoid.

The earliest reported primary tumor of the ganglion was in 1849 by R. W. Smith[22] who saw the patient in 1836. The earliest operative attempt at removal of a paratrigeminal tumor was reported by Krogius[15] in 1896. In all probability this was a malignant tumor extending upward from the nasopharynx. In 1918 Frazier[8] found 43 recorded cases of tumors involving the gasserian ganglion of which 13 were exposed surgically and only 3 deemed operable. The majority of these were also malignant. Ver Brugghen[24] as late as 1952 stated that less than 100 paratrigeminal tumors, both primary and secondary, had been reported.

Trigeminal pain may be caused by tumors in various sites with pressure in many directions.[19] Pressure on the ganglion may come from the floor of the temporal fossa, from lateralward or from the temporal lobe above, from a tumor in the sheath, or from a metastatic tumor to the ganglion. Symptoms may also arise from pressure on the root and from a growth in or exerting pressure on one of the peripheral branches. There are some authors[4,9,14] who believe that primary involvement of the sensory root results in anes-

thesia rather than any marked degree of pain, while involvement of the ganglion or peripheral branches is commonly the source of severe pain. Jefferson[12] tends to agree with them. He states that a change in sensibility with neuromas of the trigeminal root is more common than pain. He adds that most all patients have absent or decreased corneal reflexes, and of his 7 patients with neuroma of the trigeminal nerve, 4 had no pain. However, there is ample evidence that pain identical with that of true trigeminal neuralgia does occur from the pressure of tumors on the root central to the ganglion. This has been demonstrated by Bailey,[2] Dandy,[5] and many others, who have relieved tic-like pain by removal of tumors pressing on the trigeminal root. The occurrence of contralateral tic douloureux caused by tumors in the posterior fossa has also been described by Hamby,[11] and we have encountered similar cases. It is well known that one of Dandy's[5] reasons for his suboccipital approach to section of the 5th nerve for tic douloureux was the occurrence of tumors in this region as a cause of the pain. It was, of course, these and similar observations that led Taarnhøj[23] to devise his operation to decompress the trigeminal nerve for trigeminal neuralgia. It is thus obvious that tumors that involve the trigeminal nerve at any point may cause pain in the distribution of that nerve and/or anesthesia. In any event, the 3 patients described here, whose trigeminal nerves and gasserian ganglia were compressed by epidermoid tumors, all had a complete anesthesia and none complained of severe pain. The motor as well as the sensory division of the trigeminal nerve is usually paralyzed by these tumors. The muscles of mastication are, as a result, paralyzed and atrophic, and the jaw deviates toward the involved side when the mouth is opened. In addition to the trigeminal nerve, tumors in this location may involve many other structures. Two of these 3 patients showed evidence of involvement of the ipsilateral optic nerve. The motor nerves to the eye as well as the facial and auditory nerves may be affected. One of these 3 patients had a contralateral hemiplegia, evidence of compression of the cerebral peduncle. In all of these patients roentgenological examination disclosed a sharply outlined destruction of the apex of the petrous pyramid and of the floor of the temporal fossa. In all cases the onset was most insidious and the progress extremely slow. In fact, these patients were hardly conscious of their abnormalities. All 3 were between 35 and 40 years of age.

There are two tumors that the surgeon is most likely to confuse with paratrigeminal epidermoids pre-operatively. They are meningiomas and neuromas. Paratrigeminal meningiomas in general produce pain in the head and face which, while it may not be a paroxysmal shock as in trigeminal neuralgia, may be produced by stimulation of a trigger zone.[2] Davis and Martin[6] found with paratrigeminal meningiomas, and almost pathognomonic in their constancy, patches of paresthesia, anesthesia, or analgesia in the ophthalmic and maxillary areas. The radiologic pictures of meningiomas usually reveal sclerosis, bony proliferation, and destruction. It would be most unusual for a meningioma in this region to produce such extensive and sharply outlined erosion of bone as is seen with an epidermoid tumor.

Perhaps the lesion most closely resembling a paratrigeminal epidermoid is the neuroma of the trigeminal root.[12] While there may be and usually is some hypesthesia of part or all of the trigeminal area and there is occasionally a weakness of the motor 5th nerve, there is rarely a complete destruction of both the motor and sensory components with severe atrophy as is found with paratrigeminal epidermoids. With a neuroma of the trigeminal root there is papilledema in a high percentage of cases and relatively widespread involvement of the brain stem and cranial nerves, in contrast with the clinical picture of paratrigeminal epidermoids. Marked erosion of the petrous apex is also not as frequent with neuromas as with epidermoids.

The treatment of paratrigeminal tumors is surgical except in the case of widespread malignancy or the obvious case of meningioma in which invasion of bone and of important intracranial structures is so extensive as to make surgical attack hopeless, or the likelihood of aggravating the patient's condition greater than that of helping him.

In the case of an infiltrating tumor with pain caused by pressure on the trigeminal nerve considerable relief may be obtained by section of the sensory root even though the tumor cannot be removed. In such cases the nerve can usually be more readily exposed using Dandy's approach through the posterior fossa.

Paratrigeminal epidermoids may be almost completely removed with ease and with little risk to the life of the patient. Following their removal the patient may be expected to have many years of comfortable productive activity.

SUMMARY

Three cases of paratrigeminal epidermoids in relatively young persons are presented. In all instances removal of the main tumor mass was successful and the patients made good recoveries. The prognosis following removal of the contents of such tumors with only partial extirpation of their walls is excellent. The recognition and differential diagnosis of this rare lesion are discussed.

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INTERVERTEBRAL FUSION FOLLOWING LUMBAR DISC EXCISION

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ADEQUATE clinical results from several types of operations for ruptured lumbar intervertebral discs have been reported for some time.[1,3–5,9–15] Despite minor variations in technique, only three basic types of operations are performed: 1) disc evacuation plus spinal fusion by bone grafting on the laminae and dorsal spines;[1,3,13] 2) disc evacuation plus bone graft within the intervertebral space;[4,5] and 3) disc evacuation without overt attempts at fusion.[2,9,13] The latter type of procedure appears to be gaining popularity.[3,9,11,13] The need for primary fusion has long been questioned by most neurosurgeons[14] and an increasing number of orthopedic surgeons.[3,12,13] A satisfactory clinical result occurs in more than half the patients treated, regardless of the type of operation performed. There has been little experimental or clinical evidence presented that firmly establishes the value of fixation of the spine in the usual case of ruptured disc. On the contrary, most clinical evidence suggests that fusion is an unnecessary addition to surgical therapy in the usual case.[2,3,12–14]

It has been postulated previously that a common critical factor may exist in these three operative procedures that is responsible for the good results claimed in all three groups. The step common to all the procedures is evacuation of the intervertebral disc space. Experimentally, fibrous union of adjacent vertebral bodies occurs following complete evacuation of the disc space in the monkey[6,8] and in the dog.[7] Intervertebral bony fusion can be produced by curettement of the opposing cartilaginous plates at the time of complete disc evacuation.[8] This shows that fusion can occur without the intervention of grafts and that intervertebral fixation may result. The frequency of intervertebral fixation following disc evacuation and curettement of cartilage in the human is unknown. The clinical significance of intervertebral fixation so produced is likewise unknown. Therefore, the present clinical study was designed to determine the presence of intervertebral fixation following disc evacuation with cartilage curettement, and to correlate its occurrence with the clinical result obtained.

MATERIAL AND METHODS

Twenty patients were operated on for chronic recurrent lumbar intervertebral disc protrusion. All patients had had two or more episodes of com-

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plete disability from recurrent low back pain and recurrent sciatica. All patients likewise had unequivocal signs of compression of the lumbar nerve root. All patients were severely disabled by their back pain and sciatica at the time operation was advised.

In all instances no clinical improvement had occurred following at least 5 days of strict bed rest, lumbar stretching exercises, and use of analgesics. Routine pre-operative Pantopaque myelography showed the ruptured disc defect, and laminotomy and disc excision was carried out at the proper level under spinal anesthesia. Care was taken to achieve the maximum removal of the disc possible and thorough curettement of the cartilage plates in the intervertebral disc space was done. After this routine operative procedure was completed and with the wound still open, roentgen-ray marker needles were placed in the lumbar spines of the vertebrae above and below the operative site as well as in a lumbar spine one level above or below the level of the evacuated disc.

After operation, all patients remained at complete bed rest until they were able to move about in bed and hyperextend the back painlessly. Ambulation was then allowed. The time of complete bed rest varied considerably, but the average was about 1 week. The sitting position was not allowed for 2 to 3 weeks.

The patients were discharged 2 weeks following operation and were examined, in so far as possible, at approximately 6 weeks, 3–4 months, 6 months, and 1 year postoperatively. At each examination, lateral upright roentgenograms of the lumbosacral spine were taken in full flexion and full extension. Complete neurological evaluation was likewise done and a full social history was taken with particular emphasis on the type of work being done.

Of the 20 cases originally included in this series, only 10 were carried through the full 1-year follow-up as outlined. Three patients were followed through 6 months, 1 through 4 months, 2 through 2 months, and 4 had no adequate postoperative follow-up. Considerable difficulty was encountered in persuading completely well individuals to travel long distances for these check-up examinations.

Of the 16 patients seen in check-up examinations, 11 had unilateral rupture of the disc at a single level, 4 had unilateral rupture at two levels, and 1 had bilateral rupture at one level.

Roentgenological evidence of intervertebral motion was determined by measuring the change in positions of the needle markers in the lumbar spines during full lumbar extension and during full lumbar flexion. The interspinous movement, as measured by the difference in position of the markers in flexion and extension, was considered to be a reliable index of the degree of intervertebral fixation (Figs. 1, 2, and 3).

At each postoperative examination, the clinical status of the patient was evaluated primarily on the basis of: (1) the amount of pain present, and (2) the type and amount of work the patient was then doing.

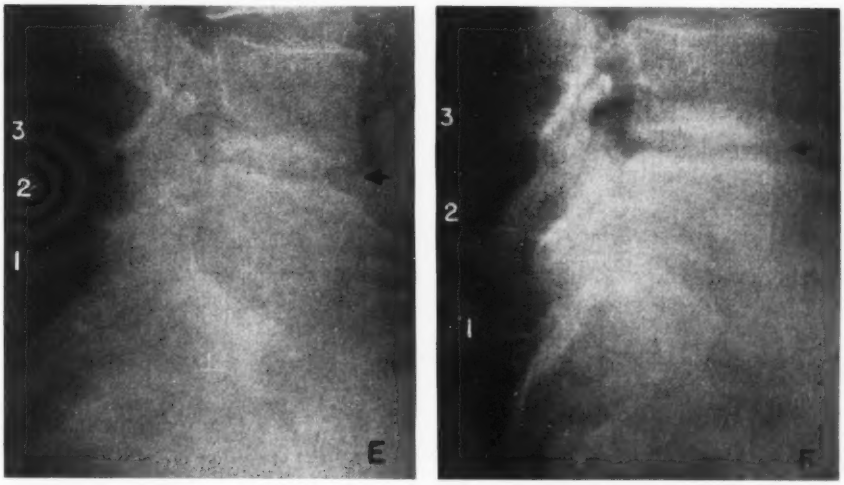

FIG. 1. *Case 4.* Extension-flexion roentgenograms 1 year after discoidectomy at L4-L5, right. Arrow indicates operative level. (E) extension; (F) flexion. Measurements between spine markers (1, 2, and 3) indicate motion at L4-L5 and L5-S1.

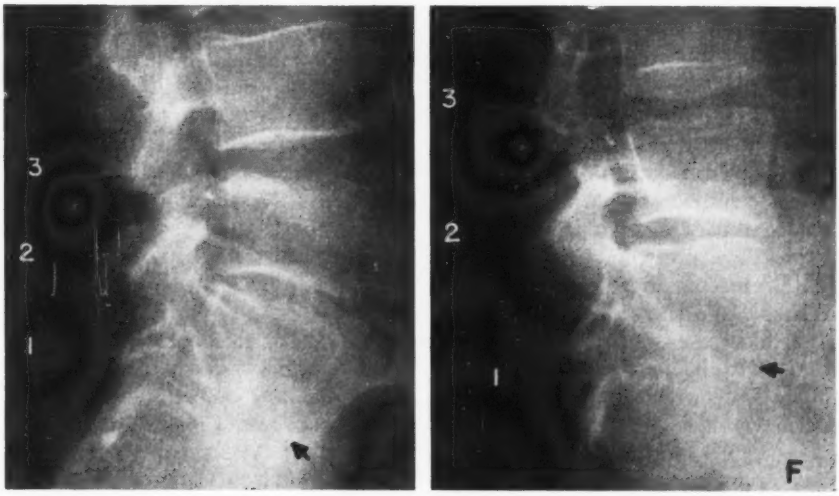

FIG. 2. *Case 7.* Extension-flexion roentgenograms 1 year after discoidectomy at L5-S1, right. Arrow indicates operative level. (E) extension; (F) flexion. Measurements between spine markers (1, 2, and 3) show motion between L4-L5 and L5-S1.

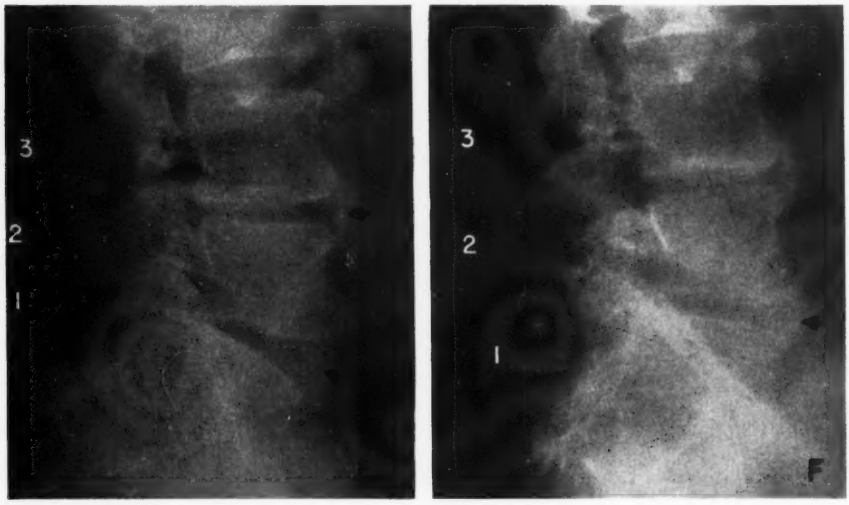

FIG. 3. *Case 12.* Extension-flexion roentgenograms 1 year after discoidectomy at L4-L5, and L5-S1, on left. Arrows indicate operative levels. (E) extension; (F) flexion. Measurements between spine markers (1, 2, and 3) indicate interspinous motion at both levels.

RESULTS

A summary of the postoperative roentgenological and clinical status of these patients is presented in Table 1, divided into 3 groups—those with a full 1-year follow-up, those with a 6-month follow-up, and those with less than a 6-month follow-up. The last follow-up examination showed a net interspinous motion by roentgen ray of at least 0.6 cm. in all cases except No. 5. Convincing intervertebral motion was present at the level of operation in 15 of the 16 patients, thereby indicating that intervertebral fixation had not occurred even by 1 year after operation (Table 1, and Figs. 1, 2, and 3).

The clinical status of all of the 16 patients at the time of their last examination was very satisfactory. Only 1 had occasional back pain and 15 of the 16 had returned to their previous work at full-time employment. Adequate information concerning the status of patient No. 14 was not available, although it is known he had returned to work.

Table 2 is a comparison of interspinous motion at the operative level and at a "normal" level in the same patient, using only those patients with a single level, unilateral disc rupture who were followed for 1 year. At the operative level, the average interspinous motion present at 4 to 6 weeks was 1.1 cm., at 6 months 1.2 cm. and at 1 year 1.2 cm. At the control level the respective figures were 1.0, 1.1, and 1.1 cm.

Table 3 shows the average interspinous motion at the various lumbar disc levels, all measurements having been taken at least 6 months after operation. The average interspinous motion at each level is compared with the other

TABLE 1

Summary of postoperative roentgenological and clinical status

Patient	Level of Operation	Time after Operation	Net Interspinous Motion (cm.) by Roentgen Ray				Clinical Status	
			L2-L3	L3-L4	L4-L5	L5-S1	Pain	Work
A. ONE-YEAR FOLLOW-UP								
1. W.F.	L4-L5 left	6 wk.			0.8	0.5	None	None
		6 mo.			1.2	0.8	None	Clerical, full time
		1 yr.			1.4	1.0	None	Clerical, full time
2. A.M.	L4-L5 right	6 wk.			0.9	0.7	None	Clerical, part time
		4 mo.			1.0	0.9	None	Clerical, full time
		6 mo.			1.1	1.0	Occasional, back	Clerical, full time
		1 yr.			0.6	0.7	Occasional, back	Clerical, full time
3. W.G.	L4-L5 right	6 wk.		1.4	1.3		None	Heavy labor, full time
		3 mo.		1.1	1.4		None	Heavy labor, full time
		6 mo.		0.8	1.0		None	Heavy labor, full time
		1 yr.		0.9	1.9		None	Heavy labor, full time
4. D.S.	L4-L5 right	6 wk.			1.5	1.0	Occasional, right calf	Clerical, full time
		3 mo.			0.4	1.1	None	Clerical, full time
		6 mo.			1.3	1.4	None	Clerical, full time
		1 yr.			0.9	1.4	None	Clerical, full time
5. G.G.	L4-L5 left	6 wk.			0.2	0.0	Occasional, left calf	Clerical, full time
		3 mo.			0.0	0.0	None	Clerical, full time
		6 mo.			0.2	0.0	None	Clerical, full time
		1 yr.			0.3	0.0	None	Clerical, full time
6. R.G.	L5-S1 right	1 mo.			0.9	1.1	None	None
		6 mo.			1.7	2.2	None	Heavy labor, full time
		1 yr.			0.8	1.7	None	Heavy labor, full time
7. H.B.	L5-S1 bilateral	6 wk.			0.3	0.3	Moderate, low back	None
		3 mo.			0.7	0.3	Moderate, low back	Light labor, full time
		6 mo.			0.0	0.2	Moderate, both hips and legs	Light labor, full time
		1 yr.			0.9	0.6	None for 4 mo.	Light labor, full time
8. R.L.	L3-L4 & L4-L5 right	6 mo.		1.9	2.2		None	Student, full time
		1 yr.		2.1	2.4		None	Student, part time clerk
9. G.T.	L4-L5 &	6 wk.			0.5	1.2	Occasional, dull, left leg	None
	L5-S1 left	3 mo.			0.8	1.1	None	Light labor, full time
		6 mo.			0.8	1.5	None	Light labor, full time
		1 yr.			0.6	1.6	None	Light labor, full time
10. G.M.	L4-L5 & L5-S1 left	6 wk.	1.9	1.9	1.5	2.1	None	None
		3 mo.	2.0	1.8	1.4	1.4	None	Heavy labor, full time
		6 mo.	2.2	1.9	2.0	1.4	None	Heavy labor, full time
		1 yr.	2.4	2.1	1.9	1.6	None	Heavy labor, full time
B. SIX-MONTH FOLLOW-UP								
11. T.D.	L4-L5 left	1 mo.			0.4	0.6	None	None
		6 mo.			0.6	0.9	None	Light labor, full time
12. E.P.	L5-S1 right	6 wk.			2.3	0.7	None	Clerical, full time
		3 mo.			0.8	0.6	None	Clerical, full time
		6 mo.			1.6	1.1	None	Clerical, full time
13. G.M.	L5-S1 right	2 mo.			0.3	0.0	None	Moderate labor, full time
		6 mo.			2.5	0.6	None	Moderate labor, full time
C. LESS THAN SIX-MONTH FOLLOW-UP								
14. M.M.	L5-S1 right	2 mo.				0.9	None	
		4 mo.				0.8	None	
15. T.W.	L5-S1 left; 1 mo. later, L4-L5 left	1 mo.			0.9		None	
		2½ mo.				0.6	None	Heavy labor, full time
16. H.S.	L5-S1 right	2 mo.			1.3	0.8	None	Clerical, full time

TABLE 2

Interspinous motion at operative and control level 6 weeks, 6 months and 1 year after operation

Patient	Operative Level			Control Level		
	4 to 6 Wk.	6 Mo.	1 Yr.	4 to 6 Wk.	6 Mo.	1 Yr.
1. W.F. (#1)	0.8	1.2	1.4	0.5	0.8	1.0
2. A.M. (#2)	0.9	1.1	0.6	0.7	1.0	0.7
3. W.G. (#3)	1.3	1.0	1.9	1.4	0.8	0.9
4. D.S. (#4)	1.5	1.3	0.9	1.0	1.4	1.4
5. G.G. (#5)	0.2	0.2	0.3	0.0	0.0	0.0
6. R.G. (#6)	1.1	2.2	1.7	0.9	1.7	0.8
7. H.B. (#7)	0.3	0.2	0.6	0.3	0.0	0.6
8. G.M. (#10)	1.5	2.0	1.9	1.9	1.9	2.1
	2.1	1.4	1.6	1.9	2.2	2.4
Averages	1.1	1.2	1.2	1.0	1.1	1.1

lumbar levels, both as a control and as an operative level. At L5-S1, the average postoperative motion in 9 cases was 1.0 cm., whereas patients who were not operated upon at this level showed a motion of 0.8 cm. This difference is probably not significant other than to show that there apparently is as good if not better interspinous motion between L5 and S1 following operation than when no operation was performed at that level.

The average interspinous motion between L4 and L5 following operation was 1.0 cm., whereas without operation the motion was 1.6 cm. At this level, the interspinous motion appeared to be significantly greater without operation than following operation.

The interspinous motion between L3 and L4 following operation was twice as much (2.1 cm.) as at the other levels. The motion at this level was 1.5 cm. when no operation had been performed. Whereas there was greater motion between L3 and L4 after operation than with no operation, the 3 cases studied are too few to emphasize.

Comparison of motion at the control levels of L5-S1, L4-L5, and L3-L4 indicates that there is twice as much motion at the upper two levels. Since the L5-S1 level was a control for operations at a higher level, and since both L4-L5 and L3-L4 levels were always controls for operations at a lower level,

TABLE 3

Average interspinous motion at various levels, operative and control

Level	Level of Operation	Control Level
L5-S1	1.0 cm. (9 cases)	0.8 cm. (5 cases)
L4-L5	1.0 cm. (9 cases)	1.6 cm. (6 cases)
L3-L4	2.1 cm. (1 case)	1.5 cm. (2 cases)
L2-L3	none done	2.4 cm. (1 case)

it would appear that in these 13 patients there was twice as much postoperative intervertebral motion above the operative level as below it.

Table 4 is a comparison of interspinous motion with operative level and relief of pain. In all cases, at least 0.2 cm. of interspinous motion was present at the operative level and in all cases relief of pain was complete, with the exception of Case 2 who had very occasional back pain. The interspinous motion at the adjacent level was 0.2 cm. or more in all cases except for Case 5 who showed no motion between L5 and S1, his control level. (It is presumed that he had achieved a spontaneous intervertebral fixation at a nonoperative level. His clinical result was excellent, however.)

In the 4 cases in which double level discoidectomies were performed (Table 4) the interspinous motion at these levels was entirely comparable in all instances to the motion present in cases in which operation was done at a single level.

Table 1 shows that 6 weeks following operation, 12 of the 16 patients

TABLE 4

Correlation of operative levels, interspinous motions, and relief of pain

Operative Level	Time Post-operative	Patient	Interspinous Motion		Relief of Pain
			Operative Level	Adjacent Level	
L4-L5	1 yr.	W.F.	+	+	Complete
		A.M.	+	+	Good
		W.G.	+	+	Complete
		D.S.	+	+	Complete
		G.G.	+	0	Complete
	6 mo.	T.D.	+	+	Complete
L5-S1	1 yr.	R.G.	+	+	Complete
	1 yr. (bilateral)	H.B.	+	+	Complete
	6 mo.	E.P.	+	+	Complete
		G.M.	+	+	Complete
	4 mo.	M.M.	+	No marker	Complete
	2 mo.	H.S.	+	+	Complete
Double levels					
L3-L4,			+		
L4-L5	1 yr.	R.L.	+	No marker	Complete
L4-L5,			+		
L5-S1	1 yr.	G.T.	+	No marker	Complete
L4-L5,			+		
L5-S1	1 yr.	G.M.	+	+	Complete
L4-L5,	6 wk.		+	No marker	
L5-S1	2½ mo.	T.W.	+	No marker	Complete

0 = no movement.
+ = 0.2 cm. or more movement.

(76 per cent) had no complaints at all of low back pain or leg pain. Of the remaining 4 patients 3 had occasional calf pain and 1 had occasional low back pain. In the 4- to 6-month follow-up, 12 of the 14 (84 per cent) were completely asymptomatic. The 2 with symptoms complained of occasional dull low back pain. Of the group followed for 1 year, 9 of the 10 (90 per cent) were completely asymptomatic. One patient had occasional back pain of mild degree.

DISCUSSION

In view of the clear-cut intervertebral motion following discoidectomy in clinical cases, it is evident that the experimental intervertebral fixation following discoidectomy in the monkey, reported by Jenkner, Foltz, and Ward,[7] was not obtained in this study.

The interspinous motion by roentgen-ray measurements at the operative level and the control levels shows very little difference throughout the periodic check-up examinations. This has been presented in Table 2 in which it may be noted that the average interspinous motion at the operative level 1 year following operation is almost the same as it was 4 to 6 weeks after operation. Likewise, at the control levels in the same patients, interspinous motion at 1 year is almost exactly the same as it was 4 to 6 weeks following operation. Apparently no progressive change in interspinous motion took place at the operative level or at the control level in this series of cases. This finding in a way was somewhat of a surprise yet might be suspected since the majority of patients having this type of disc operation show good mobility of the back clinically and are free of pain early in their postoperative course.

This proof of interspinous motion indicates that the fibrous intervertebral fusion is a flexible union and does not produce fixation. Such a fibrous union has elastic properties which do not significantly impair intervertebral motion in flexion and extension of the lumbar spine up to 1 year. Long-term studies of such cases will be helpful in supporting these data.

Evidence concerning the normal amount of interspinous motion at the levels with which we are concerned is minimal. Table 4 shows that following operation the control L3–L4 and L4–L5 interspaces provide twice as much motion in flexion-extension as does the control L5–S1 interspace. As has been pointed out, the L3–L4 and L4–L5 interspaces were in each case above the level of operation while the L5–S1 space was below the operative site. It is not known whether this represents a postoperative continuation of a normal pre-operative disparity of motion, or rather indicates a new motion ratio between these levels following operation. These levels, particularly L4–L5 and L5–S1, are by far the most common for herniation of the lumbar discs to occur but no relation of this difference in mobility to the incidence of ruptured discs is immediately apparent.

Case 5 has caused some difficulty in this series in that no motion was ever measured between L5 and S1, whereas minimal motion was present at his

operative level between L4 and L5. Possibly a spontaneous intervertebral fixation or fusion had occurred at L5–S1 prior to his disc protrusion at L4–L5. Such immobilization would tend to increase the changes in chondroitin-sulfuric acid of the cartilage in the disc, resulting in degenerative changes and favoring ultimate rupture as suggested by Woodhall.[16]

The clinical results in these 16 patients at the end of 1 year were excellent with complete relief of pain in all cases except 1 which was still classified as a good result. All of these patients had significant intervertebral motion at the operative level, and all but 1 at the control level. Clearly, intervertebral fixation after disc excision is not necessary to obtain good clinical results.

The thesis that intervertebral fixation is the common factor leading to good clinical results in the three major operative techniques for ruptured intervertebral discs is not tenable. Immobilization of the lumbar spine does not occur routinely following evacuation of the disc and curettement of cartilage. The intervertebral fibrosis which must occur, however, allows excellent mobility of the back up to 1 year following operation. It would seem, then, that the only common factor in the three major operative techniques for treating ruptured intervertebral discs is the evacuation of the ruptured intervertebral disc itself and relief of nerve root pressure.

SUMMARY

1. A series of 16 patients suffering from recurrent lumbar disc herniations were operated on by simple evacuation of the disc and curettement of cartilage. Roentgen-ray markers were placed in the lumbar spine at the level of operation and at the levels at which no operation was performed, above or below. Periodic roentgenological examination for mobility of the lumbar back and clinical evaluations were carried out for a full year in 10 of these cases, for 6 months in 3 of them, and for less than 6 months in the remaining 3.

2. All of these patients showed conclusive roentgenological evidence of good flexion-extension mobility at the intervertebral levels of operation 1 year postoperatively. This mobility was present as early as 4 to 6 weeks following operation. In no case was there development of lumbar intervertebral fixation.

3. Mobility of the lumbar back in flexion-extension at the operative level was practically the same as at the nonsurgical or control levels.

4. Normal intervertebral motion in lumbar flexion and extension was found to be twice as great between L4–L5 as it was between L5 and S1.

5. It is concluded that intervertebral fixation does not occur routinely following simple evacuation of the disc and curettement of cartilage but instead an elastic fibrous intervertebral union results which allows almost normal flexion and extension of the lumbar spine.

6. All patients in this series had excellent clinical results with complete relief of pain and return to productive work.

7. The evidence presented indirectly emphasizes again the fact that

spinal fusion and overt intervertebral fusion are not necessary as primary measures in the operative treatment of herniated lumbar intervertebral discs.

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A STUDY OF THE RESULTS OF SURGICAL TREATMENT IN 2,326 CONSECUTIVE PATIENTS WITH BRAIN TUMOR*

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TO BE asked to give the Max Peet Oration is not only a high honor but a particular pleasure. Max was my good friend. His training preceded mine with Dr. Frazier and consequently we thought along similar lines about many things. In addition to his keen clinical judgment and amazing manual dexterity in the operating room, his reputation among ornithologists was equal to his surgical standing among surgeons. Max Peet was a man of many ideas. When one's contribution to neurosurgery ranges from hypertension to poliomyelitis and from the neuralgias to head injury, it is a definite indication of an active, observant, yeasty mind. He was always intrigued by the problems presented by brain tumors. It is with great pleasure therefore that I discuss with his associates some of the results obtained in the handling of such lesions. That this material should be presented from the clinic where he obtained his earliest training appeals to me as very proper and fitting.

One advantage accruing from the survey of such a large group of brain tumors is the determination of just how much a surgeon's skill and judgment, in comparison with his luck in exposing one type of lesion rather than another, have to do with the final results. The most widely quoted statistics on brain tumor surgery are those from Cushing's clinic.[1] It must be remembered, however, in totaling these figures, that either Cushing or his equally skillful assistant, Horrax, performed all the operations. In the series here studied, the patients were operated on during the last 30 years by Frazier, Grant, and a succession of residents. The training of residents has become, over this same period, an integral and important part of the duties of any neurosurgical service. The figures of this present series go far toward showing what group of cases can, with justification, be turned over to the residents and what type of case should be handled exclusively by more experienced staff members. In many instances, the patient's situation is entirely hopeless, regardless of the experience or inexperience of the operator. But when a more favorable tumor is encountered a complete cure or at least a long survival period for the patient may depend upon expert skill at the operating table. And since the hopeless group is equal in numbers to the more favor-

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able type, it seems reasonable to assign the hopeless group to the inexperienced surgeon rather than those patients lucky enough to harbor a relatively benign tumor.

The division of the cases in the present series into 2 groups and the reference to them as brain tumors and intracranial mass lesions will further emphasize this point, albeit in a rather clumsy fashion. The brain tumors are those lesions that arise from the glial tissue, the cells of the brain itself. The intracranial mass lesions arise from any tissue within the cranium other than brain tissue—the dura mater, the glands, the blood vessels. In the first group fall all the gliomas; in the second, the fibroblastomas, pituitary tumors, hemangiomas, and all others of nonglial origin. In this series of over 2,300 cases, roughly half of the tumors were of glial origin and half fell into the intracranial mass lesion classification. Of the patients in the glial group, 146 lived 5 years or over. One hundred and fifteen of the tumors were solid or cystic fibrillary astrocytomas. There were 677 out of the 1,100 patients with intracranial mass lesions who lived over 5 years. It is obvious, therefore, when preoperative evidence indicates the presence of an intracranial mass lesion, or when a resident exposes a lesion that appears to fall into this group, that the conscientious chief of service should take over, should stand by and supervise, or should thoroughly assess the resident's skill and judgment before allowing him to assume the full responsibility for the extirpation of a potentially favorable tumor. In reviewing this group of cases, especially when the operative notes were carefully considered, it is my opinion that our operative mortality could have been reduced by 20 to 25 per cent, had a more experienced surgeon been in charge throughout. And incidentally this impressed me most obviously during the years 1920 to 1930, when I was Dr. Frazier's resident and assistant. It is essential that the resident have training and experience. But let him get his training and experience on the glial group of tumors, where no operator, no matter how gifted he may be, can accomplish very much.

The localization of a brain tumor is now a very simple matter by air studies or angiography. Notice the lack of emphasis on neurology. The history of the patient, the way in which the symptoms developed, and the over-all neurologic picture are important. But no experienced neurosurgeon would operate on these findings alone. He must have the more positive localization afforded by air studies or angiography. In this series, 37 tumors were found at autopsy to be in areas other than the operative field. Thirty-one of these cases occurred prior to 1935. Other cases that did not come to autopsy might raise the total. However, the autopsy rate on the neurosurgical service has never been less than 85 per cent. For 4 consecutive years, 1948 to 1952, it was 100 per cent, so that most of our errors in localization must have been discovered.

There were 2,326 brain tumors verified by tissue study and the survivors were all carefully followed between January 1, 1924 and January 1, 1954. In 192 cases the diagnosis was verified by autopsy. There were 2,725 opera-

tions carried out on 2,134 patients with 636 operative deaths, a total operative mortality of 23 per cent. Of these patients, 659, or 30 per cent, lived 5 years or longer. There are 528 alive to date, 418 from 5 to 30 years, 110 less than 5 years. Among these 659 patients living over 5 years, 512, or 78 per cent, returned to work. In this entire series of 2,326 patients, 1,667 died within 5 years (Tables 1 and 2).

The most favorable results follow the exposure of a nonglial tumor—an intracranial mass lesion, meningioma, acoustic neuroma, pituitary adenoma or hemangioblastoma. In our series of 659 cases in which the patients lived 5 years or longer there were 196 meningiomas, 47 acoustic neuromas, 136 pituitary tumors, 31 craniopharyngiomas, 44 hemangiomas, and 63 miscel-

TABLE 1

Statistics of entire group

Diagnosis	No. of Cases in 30 Yrs.	Survival Time after Operation (Years)						No Op. Autopsy	Postop. Deaths	No. of Ops.	Mortality
		30–25	25–20	20–15	15–10	10–5	Less 5				
Astrocytoma											
Cerebral	279	0	11	9	14	14	129	31	71	340	20%
Cerebellar	166	7	8	9	15	28	46	14	39	179	20%
	445	7	19	18	29	42	175	45	110	519	21%
Meningioma	407	14	28	34	55	65	86	14	111	520	21%
Glioblastoma	350						188	39	123	324	38%
Pituitary adenoma	206	14	26	33	29	34	20	9	41	351	11%
Metastatic tumor	196					2	113	26	55	177	31%
Medulloblastoma	112			1	2	1	54	10	44	117	37%
Acoustic neuroma	110	5	2	5	24	11	27	1	33	129	27%
Craniopharyngioma	70	2	9	6	5	9	27	3	9	95	9%
Hemangioma	64	2	10	9	11	12	10	3	7	75	9%
Ependymoma	55		1	5	1	4	24	3	17	68	25%
Oligodendroglioma	48				3	7	24	2	12	62	19%
Miscellaneous	263	2	8	16	18	19	91	37	72	288	25%
Total	2,326	46	103	127	177	206	839	192	636	2,725	23%

TABLE 2

Statistics of miscellaneous tumor group

Diagnosis	No. of Cases in 30 yrs.	Survival Time after Operation (Years)						No Op. Autopsy	Postop. Deaths	No. of Ops.
		30–25	25–20	20–15	15–10	10–5	Less 5			
Astroblastoma	62					2	43	5	12	72
Spongioblastoma unipolare	35						6	10	19	29
Neuroblastoma	25			1		1	12	3	8	22
Cholesteatoma	24	1	3	5	5	4	3	1	2	28
Osteoma	19	1	3	5	3	3	3		1	19
Tuberculoma	18			1			7	4	6	18
Papilloma	13		1	1			2	3	6	13
Pinealoma	13						2	3	8	11
Paraphyseal cyst	9			1				4	4	14
Dermoid	7			1	2	1	1	1	1	11
Arteriovenous anomaly	8				1	4	3			12
Teratoma	5					1		1	3	7
von Recklinghausen's	5			1	1	1	1		1	6
Osteochondroma	5				1	2	2			6
Gumma	5				2		1	1	1	6
Chordoma	4						3	1		5
Osteitis fibrosa cystica	4				2		2			5
Ganghoneuroma	2		1		1					4
Total	263	2	8	16	18	19	91	37	72	288

54 alive to date: 44 from 5–27 yrs.; 10 less than 5 yrs.

laneous tumors, a total of 517, or 78 per cent, in the intracranial mass lesion group. The remaining 142 cases (22 per cent) are in the glial cell series: 115 fibrillary astrocytomas, 11 ependymomas, 10 oligodendrogliomas, 4 medulloblastomas, and 2 neuroblastomas. It is readily apparent that the underlying cell type of the brain tumor always determines the eventual outcome of the case.

The fibrillary astrocytomas, 279 cerebral and 166 cerebellar, total 445, or 19 per cent of the series. A division as to position above or below the tentorium and whether the tumor was cystic or solid became necessary because the life expectancy in the various groups was so different. Of the 279 cerebral astrocytomas, 242 were solid and 37 were cystic. Of the 166 astrocytomas situated below the tentorium, 129 were solid and 37 were cystic. There were 31 cerebral and 14 cerebellar nonoperative postmortem verifications. A total of 340 operations were performed on 248 patients in the cerebral group with 71 operative deaths, a mortality of 20 per cent. In the cerebellar group, 179 operations were carried out on 152 patients with 39 fatalities, again a 20 per cent mortality. Of the 290 patients, 115 survived for over 5 years. Seventeen patients have died, 12 with cerebral astrocytomas, 8 from recurrence and 4 from intercurrent disease, and 5 with cerebellar astrocytomas, 2 from recurrence and 3 from intercurrent disease. Among the 98 alive to date, 5 years and over, 39 had cerebral tumors, 31 solid and 8 cystic, and 59 had cerebellar tumors, 39 solid and 20 cystic. Eighty-one patients were able to return to work, of whom 27 had cerebral tumors, 19 solid and 8 cystic, and 54 had cerebellar tumors, 34 solid and 20 cystic. Seventeen were disabled, all with solid tumors, 12 cerebral and 5 cerebellar. A patient with a cerebellar astrocytoma, especially if it is cystic, has an excellent chance for recovery and complete rehabilitation. If a solid cerebellar astrocytoma lies laterally in the hemisphere, so that amputation of the lobe and complete removal can be accomplished, the prognosis should be favorable. Amputation of one-half to two-thirds of the cerebellar hemisphere leaves little or no neurologic deficit after 6 months have passed. The midline cerebellar astrocytomas, encountered usually in children, have a much graver prognosis. However, with careful surgery much can be accomplished. Three patients are alive and well over 20 years, 3 for 15 years, 5 over 10 years, 1 over 9 years and 3 over 8 years. One died in an accident, 11 years after a complete extirpation of a midline astrocytoma. Three died of recurrence after a partial removal 14, 13, and 10 years later. Of these 19 patients living 8 years or longer, 15 had no neurologic deficit.

Among the 39 patients with cerebral astrocytomas alive to date, 27 were able to return to work. In these 39 patients, the location of the tumor in 15 instances was in the temporal lobe, in 10 the frontal, in 9 the parietal, and in 5 the occipital lobe. In every case the right hemisphere was involved. A complete lobectomy behind the tumor was carried out. The 12 patients unable to work following surgery were hemiparetic in 8 instances (6 had

some weakness preoperatively) and in 4 convulsive attacks developed. In 3 of these 4 cases the focal convulsions had helped to localize the lesion.

Damage to a cerebral hemisphere in an attempt at complete removal of an astrocytoma may be much more disastrous to the patient than a similar injury to the cerebellum. But since cerebral lobectomy seems to be the only way to excise completely an astrocytoma with any certainty, such an attempt should always be made.

The remaining glial tumors may be briefly discussed. No patient with a glioblastoma multiforme survived over 5 years. Ten among 50 operative survivors harboring an oligodendroglioma lived 5 years or more and then died of a recurrence. Two among 62 patients with astroblastomas lived over 5 years and recurrence killed them. Four among 58 patients with medulloblastomas lived over 5 years. In these 4 cases, the age at operation was over 15 years in each instance. Irradiation therapy was vigorously employed. That there is a difference in potentiality for recurrence in the cells of medulloblastomas appearing in children over 15 or in young adults, as compared to similar tumors removed from children under 15, seems clear. Complete surgical extirpation of a medulloblastoma is impossible, yet 3 of these older patients are alive to date, 17, 12 and 9 years postoperatively. A fourth died of recurrence after 12 years. A glance at Table 2 will show the unfortunate situation faced by surgeon and patient when the rarer types of glial tumors are encountered.

The meningiomas have always been presumed to be a favorable group of tumors. But this applies only to those meningiomas situated over the convexity of the brain and not broadly attached to the falx. Any meningioma lying at the base of the brain, from the olfactory groove to the foramen magnum, presents a very difficult and tantalizing problem. The unfortunate fact is that when a neurosurgeon encounters a meningioma he is under great pressure to attempt total extirpation and thereby cure the patient. Is total extirpation possible or proper, however, when it may result in carotid hemorrhage or damage to hypothalamus or brain stem with subsequent severe neurologic deficit or death?

The meningiomas numbered 407, or 17 per cent, of the total series of 2,326 verified tumors. In 14 cases, 4 convexity and 10 basilar tumors, the lesion was verified at autopsy. There were 520 operations carried out on 393 patients with 111 fatalities, an operative mortality of 21 per cent. Of these patients, 196 lived 5 years or over.

The convexity tumors are the most numerous, produce striking symptoms of convulsions, hemiparesis, and sensory disturbances, and present the easier surgical problem. Every effort should be made to extirpate completely a tumor in this region. The frontal basilar group are accompanied by relatively little neurologic evidence of their presence—for the most part mental changes, proptosis of one eye, a visual field deficit, or ocular muscle paresis. Local changes in the bone on roentgen-ray studies may be the only evidence

of the presence of a subfrontal tumor. Since these patients are not seriously crippled by the lesion, an attempt at complete removal should not be carried to the point at which the patient suffers more from the surgeon's efforts to remove the tumor than he did from the tumor itself. The subtentorial tumors may cause increased intracranial pressure, cranial nerve and cerebellar symptoms rather promptly. A definite effort should be made to remove these lesions completely. A meningioma in the posterior fossa is often easier and safer to extirpate than an acoustic neuroma, since it is usually situated more laterally and is therefore less likely to be adherent to the brain stem. The 7th and 8th nerves may be injured, but the cerebellar symptoms disappear quickly if pressure is relieved, and remarkably little permanent damage is noted.

A study of the postoperative fatalities in these cases brings into clear relief many of the problems involved. Nineteen patients (5 with convexity and 14 with basilar tumors) died of postoperative shock. By this term is meant a fatality within 24 hours, obviously caused by operative trauma alone, with deep stupor, a failure of pulse and blood pressure, a rise in respiratory rate, and hyperthermia. The fact that only 5 of the 59 deaths in the convexity tumor group were attributed to that cause, whereas 14 of the 52 basilar tumor fatalities fell into that category, is significant. Injury to the carotid vessels, hypothalamus, or brain stem in the basilar group is a disastrous complication. Fatal postoperative shock following extirpation of a brain tumor is not caused by loss of blood alone. Brain damage will produce it without severe bleeding. The judgment of the operator has always been at fault.

Prevention of postoperative shock depends on two factors: first, the experience of the surgeon in knowing just how far he can carry the procedure with safety and second, an adequate supply of blood. And the presence of ample blood for transfusion is not always an unmixed blessing, for the surgeon may rely too much on replacement to cover his errors in judgment. An operator often attempts to remove a tumor in one stage and then, to his chagrin, discovers that a replacement, even greater than the amount of blood lost, will not rescue the patient.

Exposure of the tumor brings up a critical decision—will the patient withstand further operative trauma involved in the removal of a tumor of this size in this position? Should extirpation be attempted, the maneuver may well be accompanied by further loss of blood and brain damage with consequent drop in pulse pressure and volume. The operator may find himself with a tumor half in and half out of the wound and be compelled to complete extirpation in order to close the wound. The consequent bleeding and brain damage may produce a state of shock, which often does not respond to transfusion, drugs, or any methods for relief. If any doubt exists after exposure of the lesion and before any determined effort to remove it has been made, close the wound with proper supportive measures. These tumors are benign and slow-growing. Nothing serious can happen to the patient if final removal of the lesion is postponed for a week or two. In han-

dling a subfrontal meningioma remember that these tumors usually produce minor neurologic defects and grow very slowly. Curiously enough, tumors accompanied by the most obvious invasion of bone are much smaller in intracranial content than lesions accompanied by a minimal bone change. If a tumor produces marked thickening of the orbital plate and sphenoid ridge with few or no severe neurologic deficits, no attempt should be made to extirpate it. The surgeon must take every precaution to minimize operative neurologic damage in attacking a subfrontal tumor. Few patients appreciate being told that their tumor has been completely removed when they face living their normal life span with a partial or complete hemiplegia, which was not their preoperative complaint.

There were 238 tumors situated on the convexity of the skull. In 150 cases complete and in 88 partial removal was possible. In this group of 238 cases, 306 operative procedures were carried out, with 58 deaths, 32 after complete and 26 following a partial removal, an operative mortality of 19 per cent. Of the 117 patients who survived complete removal, 80 were able to return to work. Thirty-seven were disabled. Among the 62 patients in whom only a partial removal could be accomplished, 20 are working 5 years or longer, 31 are disabled 5 years or longer, and 11 died of recurrence after an average of 8 plus years. Three of these worked until a recurrence became apparent; 8 were disabled until they died. So that of the 177 surviving patients with convexity tumors, 103 were able to return to work postoperatively. Eighty of this group had a complete removal of the tumor and can be considered as cured.

The meningiomas of the convexity of the skull, therefore, although they are presumed to be favorable tumors, benign and readily removed since their position permits accessibility, have not, in our hands at least, been attacked with any notable success.

If this statement is correct for meningiomas of the convexity, its truth is more apparent when the 155 basilar meningiomas are considered. In the latter group 214 operations were performed with 52 deaths, an operative mortality of 24 per cent. Of the 155 tumors 47 were totally extirpated. Twenty-seven of the patients were able to return to work, and 20 were crippled. In 56 instances a partial removal was all that could be accomplished with safety. Twenty-six of the patients returned to work, and 30 were disabled. Of this group, 26 had died of recurrence after an average of 6 plus years' survival. Of these 155 patients with basilar meningiomas, 103 survived the operative procedure, 53 returned to work, and 50 were unable to return to work, principally because of visual loss, hemiparesis or convulsive episodes.

The acoustic neuromas are another example of a benign tumor in a position surrounded by surgical hazards. The posterior fossa is a cramped, narrow terrain. The tumor commonly may be found adherent to the brain stem and to the vagal complex of nerves. Dandy[2] showed in 1922 that complete extirpation could be carried out with skill, patience and luck. Ever since that

time the neurosurgeon has been harassed by the knowledge that a recurrence will not take place if he can accomplish complete extirpation. Consequently he pushes himself into an all-or-none attitude. Judging by our records, too much has been attempted too often. The facial nerve, in our experience, has never been saved. A facial-hypoglossal anastomosis has produced a fair measure of facial symmetry.

The crux of the situation in an attack upon an angle tumor is the degree of its adherence to the adjacent brain stem. If this adhesion is close, if a noticeable amount of bleeding follows separation or attempted separation requiring implantation of muscle, gelfoam or clips along the stem, the immediate result may seem successful, but in all probability edema of the brain stem will develop and the patient will die on the 3rd or 4th postoperative day. Generally speaking, complete extirpation should be pushed to the limit of safety in younger patients below 50 years of age. Among 110 patients, 21 with complete removals are alive, and 17 are functioning normally except for expressional difficulties helped by a hypoglossal-facial anastomosis. Another patient with complete removal died of heart disease after 25 years of a useful life. A second died of cervical carcinoma after 7 years of normal existence. Three patients with complete removals were badly crippled and died within a year as the result of their postoperative neurologic deficit. Thirty-six patients with partial removals lived an average of $8\frac{1}{2}$ years following surgery and 17 were able to carry on a useful occupation for a time at least. Thirty-five patients died postoperatively, 26 after a partial and 9 after a complete extirpation. One patient died unoperated upon and the tumor was verified at autopsy. One death following complete and 1 following a partial extirpation resulted from attempts to remove bilateral lesions. The over-all operative mortality was 27 per cent.

The glandular tumors within the cranium, and here the pituitary lesions only are under consideration, are on the whole a most satisfactory group. There were 206 patients with pituitary adenomas, most of the chromophobe type. Of these, 41 died postoperatively; 9 cases were verified at autopsy. There were 351 operations performed on 195 patients, with 41 fatalities, an operative mortality of 11 per cent. Of these, 136 lived 5 years or longer, and 111 were able to return to work. In 19 the vision was poor preoperatively and did not return sufficiently to permit any gainful occupation; in 4 convulsive attacks developed, and in 2 a crippling hemiplegia followed surgical intervention. Irradiation therapy is not used routinely pre- or postoperatively. If visual fields show evidence of recurrence, then the effect of roentgen rays is determined. But the experience in this clinic leads us to rely upon evacuation of the adenoma by surgical means rather than to temporize with roentgen-ray therapy. The use of ACTH and cortisone is an extremely valuable adjunct and should always be employed pre- and postoperatively in the advanced hypopituitary subject to prevent total glandular collapse after surgery.

Seventy craniopharyngiomas are included in this series. There were 95 operations performed on 66 patients, with 8 deaths, a 9 per cent mortality. Four cases were verified at autopsy. Of the patients operated upon, 31 survived 5 years or longer, and 20 were able to return to work. Of the 11 crippled patients, 3 had a hemiparesis, 6 ocular difficulties, and 2 convulsive attacks. With this lesion ACTH and cortisone will rescue the bad pre- and postoperative risks.

The hemangiomas, unfortunately few in number, have a relatively satisfactory prognosis especially when they are cystic and subtentorial. Forty-two were cerebellar, and 22 were cerebral. There were 3 autopsy verifications, and 7 operative deaths. Of the 54 patients who survived operation, 44 lived over 5 years, 27 with cerebellar and 17 with cerebral lesions. Twenty-three of the cerebellar group returned to work. In the remaining 4, visual difficulties intervened. Twelve of the patients with cerebral lesions were cured. Five were crippled by hemiparesis (3) or convulsive attacks (2).

The final nonglial group to be considered are the metastatic lesions. Among 196 patients, 2 lived over 5 years and returned to work. Of the 113 living less than 5 years, 31 were able to support themselves for a year or two before the recurrence appeared. It is in the operative management of tumors of the metastatic group that the resident staff should be allowed the greatest liberty in handling the problems that arise.

In summary, the future for a patient harboring a brain tumor seems quite grim. In this series one-half (1,169) of the tumors were of glial origin. The astrocytomas comprise about 40 per cent of the glial group. Only the solid or cystic astrocytomas of the fibrillary type, situated either below the tentorium or in an inactive part of either cerebral hemisphere, thus permitting a lobectomy behind the tumor, afford the patient any chance of a 5-year survival. Among 1,169 patients with glial tumors, 149, or 15 per cent, lived over 5 years. In these 149 cases 115, or 70 per cent, of the tumors were fibrillary astrocytomas.

In the nonglial group of 1,157 cases, 510, or 44 per cent, of the patients lived over 5 years. There were 196 meningiomas, and 136 pituitary adenomas. But the meningiomas outnumbered the pituitary group almost 2 to 1. The results in the cases of meningiomas are disappointing, contrary to our expectations. Olivecrona's[3] recently published splendid figures show what truly expert surgery can accomplish with the meningiomas of the cranial vault. Unfortunately only about a half of these tumors are thus favorably situated. The meningiomas of the base of the skull can pose many completely insuperable surgical problems. Furthermore, in 17 of our cases the tumor recurred with great rapidity in spite of an apparently total extirpation. These tumors may well be likened to the little girl in the nursery rhyme that when they are good they are very very good but when they are bad they are horrid.

The pituitary group, adenomas and craniopharyngiomas, stand out in

unexpected relief. And with the proper use of cortisone and ACTH the records can be improved.

The review of this series of tumors simply confirms two impressions. The type of tumor found determines the success or lack of success the surgeon can expect. The nonglial group of lesions provide far and away the more satisfactory results. When a tumor of this type is encountered, the very best and most experienced team the clinic can assemble should be put to work on it.

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BENIGN CYSTS OF THE BRAIN SIMULATING BRAIN TUMOR

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IN GENERAL the term "benign cyst of the brain" would be regarded as indicating a porencephalic or arachnoidal cyst. Although there is no complete agreement as to the definition of these terms or as to the etiology of these conditions, it is generally believed that the term "porencephalic cyst" refers to a congenital condition in which a cystic cavity is created by a defect in the brain. Such cysts usually communicate either with the ventricular system or with the intracranial subarachnoid spaces.[5,6,11,12,15–17] Occasionally a cystic cavity is seen that is separated from the ventricular system or the subarachnoid space by a thin membrane. Such cavities may be distinguished by the terms: incomplete porencephaly, pseudoporencephaly or closed porencephaly.[16] In the main, however, any symptoms of cerebral involvement presented by patients with porencephalic cysts arise as the result of the cerebral defect and not because of the cyst, which is purely passive and secondary development. The symptoms most commonly seen in such cases are hemiparesis and convulsions. Arachnoidal cysts may arise secondary to an infectious or inflammatory process in the leptomeninx, or they may be congenital or occur following severe cerebral trauma.[2,3,8,9,14,19,20] If symptoms of cerebral involvement are present with such cysts, convulsions are the most common.

Here we are concerned with a group of cysts that do not fall into either of the above categories, neither are they neoplastic in origin. These cysts lay within the brain substance and did not communicate either with the ventricular system or the subarachnoid space. They gave rise to symptoms and signs of increased intracranial pressure and were associated with other manifestations which were similar to those commonly associated with intracranial neoplasms. Two of these cysts lay in the cerebral hemisphere and one was in the cerebellum.

CASE REPORTS

Case 1. I.B., a 25-year-old married female, was first seen at the University of Chicago Clinics in 1931, complaining of convulsive seizures, tremor of the right hand and headaches of 1 month's duration. She had had several convulsions in childhood, and they recurred in 1923 following the birth of her child. They had then again subsided to recur in 1930 and to continue at intervals of every 2 or 3 months.

Examination. On May 27, 1931, positive neurological findings were a right hemi-

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paresis, a right hemihypesthesia, and a right lower quadrantic homonymous hemianopsia. Lumbar puncture revealed nothing abnormal. The initial pressure was 160 mm. of water. The fluid was clear and colorless. It contained 3 white blood cells. There was a trace of globulin on Pandy's test. The Wassermann reaction was negative and the colloidal gold curve was 0000000000.

She was placed on anticonvulsant medication but when she returned in February

FIG. 1. *Case 1.* Ventriculogram showing the left lateral ventricle separated from an enormous cyst by a thin membrane. There is also a pronounced shift of the entire ventricular system from left to right.

1932, her convulsions were more frequent and her headaches more severe. The findings on examination were unchanged.

Operation. On Feb. 24, 1932, ventriculography was done. Through burr holes in the occipital region blunt cannulae were inserted into the region of both lateral ventricles. In all, 75 cc. of clear colorless fluid were removed and replaced with air. The ventriculogram revealed an enormous cyst in the left temporoparietal region. It measured 10×6.5 cm. in the lateral roentgenograms and 7×10 cm. in the anteroposterior films. The cyst was separated from the left lateral ventricle by a thin membrane (Fig. 1) and the ventricular system was displaced toward the right. In the lateral view (Fig. 2) the lateral ventricle was lifted upward and forward and again was separated from the cystic cavity by a layer of cerebral tissue. Until these films were seen it was not realized that the cannula on the left side had entered a cystic cavity. Fortunately the fluid from the two needles had been collected separately. That from the right needle (right lateral ventricle) contained 21.8 mg. of protein per 100 cc. That from the left needle (cystic cavity) contained 95.5 mg. per cent.

On Feb. 29, 1932, additional roentgenograms of the skull were made. The cystic

cavity was still well outlined but there was no air in the ventricular system. This suggested that the two did not communicate.

Subsequent Course. Following drainage of this cyst the patient's condition was greatly improved and she considered herself well until September 1936, when convulsions, headaches, a right homonymous hemianopsia, and loss of memory recurred.

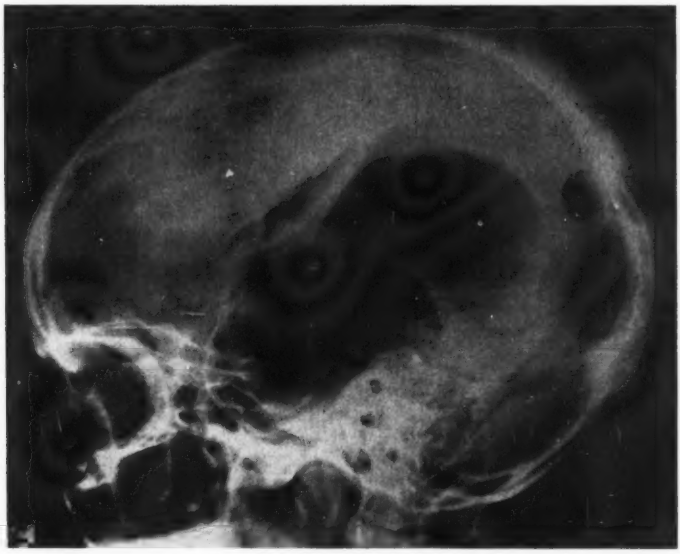


FIG. 2. *Case 1.* Lateral ventriculogram showing the lateral ventricle lifted upward and forward and clearly separated from the large cystic cavity by a layer of cerebral tissue.

On Nov. 12, 1936, a needle was inserted through the old burr hole on the left side and 130 cc. of yellow fluid were removed from the cystic cavity, and 120 cc. of air were injected. Roentgenograms again demonstrated the cystic cavity which had not changed during the more than 4 years that had elapsed (Fig. 3). There was no air in the ventricular system and it seemed very likely that the cystic cavity did not communicate with the ventricular system or the subarachnoid space. The cystic fluid contained 96.2 mg. per cent of protein. However, the patient became worse after this procedure, and aphasia, severe headaches, and stupor developed. The cyst was again evacuated. The advisability of making a craniotomy and establishing a communication between the cyst and the lateral ventricle was considered. However, as she promptly recovered this was not necessary. When seen in January 1937, her speech was somewhat slow and the defect in the visual field was still present, but there was no definite aphasia and she had had no convulsions.

Her condition remained satisfactory until Mar. 17, 1944, when convulsions, visual hallucinations, and headaches appeared. Then she was admitted to The Chicago Memorial Hospital on Mar. 24, 1944.

Neurological examinations again revealed a right hemiparesis and a defect in the right homonymous visual fields.

On Mar. 25, 1944, a needle was inserted through the burr hole in the left occipital region. The cystic fluid was under a pressure of 150 mm. of water. About 25 cc. of fluid were removed and all of her complaints disappeared in a few days, although the abnormal neurological findings persisted.

The patient returned in October 1949 with complaints of numbness of the right hand, generalized convulsions, and squinting of the right eye, and again in

FIG. 3. *Case 1.* An enormous cyst which has not changed in size during the more than 4 years that elapsed since it was first visualized. There is no evidence of any communication between it and the lateral ventricle or the subarachnoid space.

October 1952 because of headaches. Examination on both occasions disclosed a minimal right hemiparesis, a hemihypesthesia, and a right homonymous hemianopsia. Roentgenograms of the skull revealed nothing new. The cyst was not tapped. She was reassured and soon resumed her full activities. The patient was considered well when last seen on Nov. 26, 1954, except for the same minimal right hemiparesis and hemihypesthesia and the defect in the visual fields.

Comment. It seems likely that the lesion in this case has existed since her childhood. The etiology of the cyst is unknown. Neither is it clear why simple tapping of this large cyst which does not communicate with either the ventricle or subarachnoid space has sufficed to keep this woman comfortable and nearly symptom-free for over 24 years. We have always been ready to establish a communication with the ventricular system but that has never been necessary.

Except for the location of the cystic cavity in the frontal region the following case is remarkably similar. Here, however, simple evacuation of the cyst did not suffice but establishing a communication with the ventricular system has provided relief which is apparently permanent.

Case 2. C.H., a 26-year-old housewife, was admitted to the neurosurgical service at The Chicago Memorial Hospital on Apr. 5, 1942, because of generalized clonic convulsions.

About 1 year previously, during her sixth month of pregnancy, there suddenly developed several major and minor seizures without any evidence of toxemia, followed by the premature birth of the child, who died after 1 day. Subsequently the patient complained of diplopia, headaches, and transient spells of blindness. Two weeks before admission, the headaches grew more severe and the patient had nausea and vomiting. Examination in another institution revealed papilloedema and an enlarged sella turcica, and the patient was started on roentgen-ray treatment. She grew worse, however, and on Apr. 5, 1942 she suffered from three generalized convulsions. She was then transferred to The Chicago Memorial Hospital.

Examination. On admission, she was semicomatose. Papilloedema was present bilaterally. There were retinal hemorrhages and some secondary optic atrophy. There was a left lower facial weakness. Roentgenograms of the skull revealed decalcification of the dorsum sellae and thinning of the floor of the sella turcica.

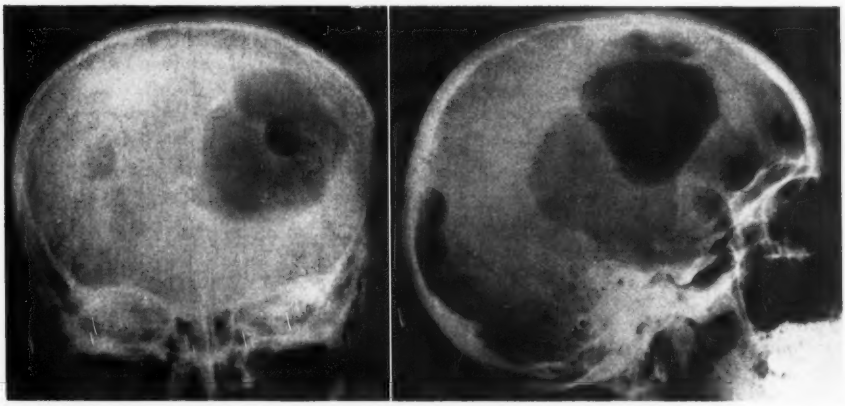
Operation. On Apr. 6, 1942, ventriculography was performed. In spite of repeated attempts, the right lateral ventricle was not entered. The left lateral ventricle was entered with considerable difficulty and approximately 10 cc. of fluid were removed. No more could be obtained. Ventriculograms revealed air only in the left lateral ventricle. Its anterior horn was displaced far to the left. A very large infiltrating glioma of the right frontal lobe was suspected.

The patient was returned to the operating room but because of her poor condition it was thought wise to be content with a decompression, and not to attempt to expose the tumor.

Through a linear incision a large bony defect was made in the right temporal region. The dura mater was very tense. A blunt cannula was inserted into the cerebral substance. When the needle was passed forward a large cystic cavity was entered. About 110 cc. of pinkish fluid under great pressure escaped. Microscopic examination of the fluid revealed a moderate number of very crenated red blood cells. When the fluid was centrifuged, the supernatant fluid was faintly yellow. However, it had the consistency of cerebrospinal fluid. The cystic fluid contained 38 mg. of protein per 100 cc. in contrast with the ventricular fluid which contained 26 mg. per cent. After the fluid had been removed, 20 cc. of air were injected into the cystic cavity and additional roentgenograms were made. The cortex of the temporal lobe appeared normal. The roentgenograms revealed a large cystic cavity in the posterior part of the right frontal lobe (Figs. 4 and 5). There was no evidence that the cyst communicated with the ventricular system, and it had every appearance of lying within the cerebral substance.

Because of our experience with I.B. (Case 1) it was hoped that mere evacuation of the cyst would suffice. Following this operation, the patient became more alert and had no headaches. But about once every 3 days it was necessary to aspirate 20 to 40 cc. of bloody fluid from the cyst, and it soon became obvious that something further would have to be done.

2nd Operation. On Apr. 15, 1942, an osteoplastic flap was reflected in the right frontal region. In the upper part of the exposed cortex the wall of the cyst was thin and transparent. An opening was made through this thin cortex into the cavity. The cyst was cone-shaped and extended into the white matter of the frontal lobe. There was no evidence of any tumor or vascular abnormality anywhere in the cyst or its wall. A blunt cannula was inserted through the floor of the cyst into the anterior horn of the lateral ventricle. Following down this needle-track tissue was resected, making an opening 1.5 cm. in diameter connecting the cystic cavity and the lateral ventricle.



FIGS. 4 and 5. *Case 2.* (*Left*) Large cystic cavity lying within the cerebral substance. (*Right*) Lateral view showing the large cystic cavity in the posterior part of the right frontal lobe. There is no communication with the ventricular system or subarachnoid space.

Postoperative Course. The patient made a good recovery. At the time of discharge (19 days postoperative), she had a slight left hemiparesis, slight diminution of vision, and minimal papilloedema. These soon subsided completely.

The patient was seen at intervals and considered well until over 11 years later. On Dec. 31, 1953, she had a generalized convulsive seizure. Neurological findings were normal. Roentgenograms of the skull revealed only the old bone flap and a parietotemporal decompression. An electroencephalogram showed a spike seizure focus in the right parietal area spreading to the right temporal and left parietal areas as well as minimal slowing in the right temporal area. She was then placed on anticonvulsant medication and has remained well ever since except for an occasional minor seizure.

*Case 3.** M.H., a 34-year-old housewife, was admitted to the University of Chicago Clinics on Oct. 16, 1936, complaining of severe headaches of intermittent nature and of blurring of vision.

The patient was perfectly well until August 1935, when occipital headaches developed. These headaches were transitory and not too severe. About the middle of June 1936, 4 months prior to admission, the headaches suddenly became much

* This case was briefly reported by Bucy[2] previously.

worse and almost continuous. In September 1936, she first noticed blurring of vision. In an effort to alleviate the condition, the patient had her teeth removed, her nasal septum extirpated, and had bought glasses, all to no avail.

The family history revealed that the patient's sister had had severe headaches, which began at the age of 20 years and occurred every 3 to 4 weeks, with occasional nausea, vomiting, and blurring of vision. At the age of 35 years, following a "stroke" and paralysis of the right side, she died.

Examination. On admission, positive neurological findings were bilateral papilloedema of 3D., bilaterally positive Babinski sign, nystagmus on looking to the left, and adiadochokinesis in both upper extremities. The spinal fluid was under a pressure of 420 mm. of water.

Ventriculography on Oct. 20, 1936 disclosed symmetrically dilated lateral and third ventricles. A cerebellar tumor was suspected.

Operation. A suboccipital craniotomy was made on Oct. 22, 1936, and a cyst was found occupying the position of the cerebellar vermis (Fig. 6). The cyst contained 45 to 60 cc. of clear, colorless fluid similar to cerebrospinal fluid. The cyst did not communicate with the fourth ventricle or the subarachnoid space. The cyst was evacuated and its posterior wall was removed.

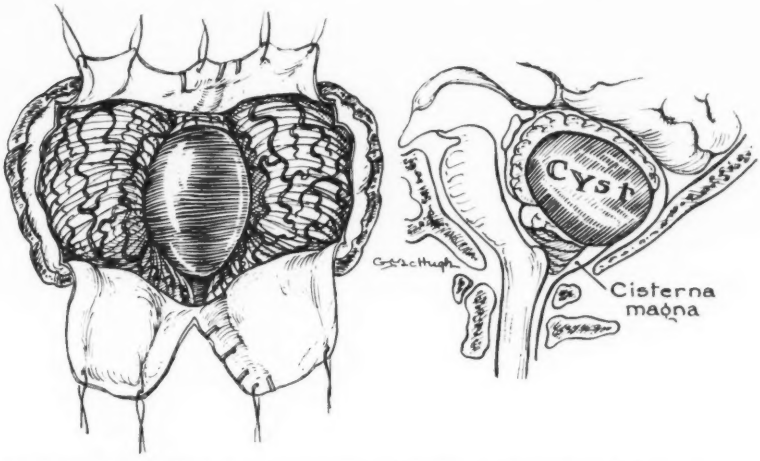


FIG. 6. *Case 3.* Illustration of the appearance and location of the cerebellar cyst seen at operation. It was entirely separate from the fourth ventricle and the cisterna magna.

Microscopic Examination. The wall of the cyst was composed of a fibrous membrane considerably thicker than the normal arachnoid membrane and at one point there was a small tuft of choroid plexus with papillae smaller than those seen in a normal choroid plexus (Fig. 7). There seemed little doubt but that we were dealing here with a congenital anomaly and that the cyst had been filled by the secretion of this small tuft of choroid plexus.

Postoperative Course. All symptoms were completely relieved and she made a complete recovery and remained well. When last seen on Dec. 20, 1955, she had no neurological symptoms and was leading a normal life.

FIG. 7. *Case 3.* Photomicrograph of the cyst wall. A fibrous membrane and a small tuft of choroid plexus are seen. Hematoxylin and eosin, ×32.

Comment. Kahn, *et al.*[8] reported an almost identical case in a 62-year-old man. Trowbridge and French[20] reported a similar case except that no tuft of choroid plexus was found. They also referred to similar cases reported by Craig,[3] Kaplan,[9] and Thompson.[19] They too concluded that these resulted from congenital anomalies, and were inclined to discount inflammation (Craig) and trauma (Thompson) as etiological factors. Our case raises one further point for speculation. In view of the history of our patient's sister is it not also possible that such cysts may be familial or hereditary?

DISCUSSION

These 3 patients presented symptoms simulating those of tumors of the brain. It is unlikely that such cases will ever be correctly recognized except on ventriculography (when the cystic cavity is entered directly), at operation, or at autopsy. The results of treatment in these cases have been most gratifying and it seems likely that such will usually prove to be the case. Surgical intervention certainly will usually be required in such cases. It is not to be expected that simple evacuation will often prove to be adequate treatment as it was in our Case 1. In most instances it will probably be necessary either to create a communication between the cystic cavity and the ventricular system, as we did in Case 2, or between the cystic cavity and the subarachnoid space (cisterna magna) as we did in Case 3. Others have advocated the complete removal of the cyst and its wall by making a lobectomy.[6] Such may be necessary occasionally, particularly when the cyst is

associated with extensive scarring of the neighboring brain and convulsions are the principal symptom. However, in most instances such treatment will not only be unnecessary but definitely undesirable. In addition to Case 2 reported here, Drew and Grant[6] and Miller[13] have reported other cases in which good results have followed the making of a communication between the cystic cavity and the ventricular system. Their patients were all followed for about 8 years.

Cases similar to our Cases 1 and 2, in which symptoms simulating those of intracranial tumor were associated with benign non-neoplastic intracerebral cysts, have been reported by Love and Groff,[11] Pendergrass and Perryman,[16] Drew and Grant,[6] and Miller.[13] However, no one has been able to elicit any definite evidence that explains the development of increased intracranial pressure in these cases. In our Case 1, the patient suffered from increasing headaches but there were no objective signs of intracranial hypertension. In Case 2, however, the intracranial pressure was seriously increased. In this instance there was evidence of hemorrhage into the cyst. The fluid was pink and contained many crenated erythrocytes. When the fluid was centrifuged it was somewhat yellow. In Case 1 the fluid was quite xanthochromic when it was aspirated some 4 years after the first operation. Both of these developments indicate that hemorrhage can occur into these cysts and that such a development may be responsible for an acute aggravation of symptoms and the appearance of signs of increased intracranial pressure.

Intracerebral cysts of this type must be differentiated from parasitic cysts and neoplastic cysts. The only parasitic cyst with which these benign intracerebral cysts are likely to be confused are the hydatid or Echinococcus cysts. Fortunately such parasitic cysts are so rare in the United States that the differentiation will seldom have to be made. In any instance, however, simple inspection will serve to distinguish between them. The hydatid cyst has a definite membrane of its own,[1] whereas there is no distinct membrane with the cysts described here. In cases of doubt, however, the surgeon should never attempt to make the differentiation by aspirating the contents of the cyst. The hydatid cysts contain numerous hydatids which are apt to be scattered if the cyst is punctured, giving rise to many daughter cysts in other parts of the brain. Differentiation from neoplastic cysts may not be possible until the cyst is actually exposed. However, the fluid within neoplastic cysts is usually distinctly xanthochromic and has a much higher protein content than was found in the cysts reported here.

Two other points here attract one's interest. In both Cases 1 and 2 the symptoms appear to have been either precipitated or aggravated by pregnancy. Why this is true is not apparent, but similar observations have been made regarding the symptoms of intracranial tumors.[4,10]

In Case 1 convulsions occurred at intervals of many years, although there have been none now since 1949. In Case 2 convulsions also occurred prior to the operation. Following the operation these ceased and did not recur until over 11 years later. These cases indicate the difficulty in assessing the sur-

gical treatment of epilepsy and in concluding that any particular form of surgical intervention has abolished convulsive seizures. The observations are also in keeping with those of Foerster and Penfield[7] who reported that post-traumatic epilepsy may be delayed for as long as 14 years after the injury.

Intracerebellar Cysts. There appears to be no group of symptoms typical of a benign cyst of the cerebellum. The symptoms are merely those of increased intracranial pressure with mild evidence of cerebellar dysfunction. It is only natural, therefore, that they will usually be mistaken for an intracerebellar neoplasm. In most instances the localizing diagnosis will have to be made by ventriculography. At operation the surgeon must distinguish these cerebellar cysts from instances of atresia of the foramina of Magendie and Luschka. As Taggart and Walker[18] have shown, such malformation of these foramina results in an obstructive hydrocephalus with a tremendous dilatation of the fourth ventricle. The dilated fourth ventricle often almost completely fills the posterior fossa, severely compressing the cerebellar hemispheres into the lateral angles of the fossa. Symptoms of atresia of the foramina of Magendie and Luschka usually appear early in childhood, whereas these benign cerebellar cysts give rise to symptoms in adult life. In the case reported by Kahn and his associates[8] the patient was 62 years of age. Furthermore, the results of surgical therapy have been poor in patients with atresia of the foramina of Magendie and Luschka whereas it is to be expected that the results will be much better with such benign cysts of the cerebellum as we have reported here.

SUMMARY

Three cases of benign cysts of the brain are reported. Two were in the cerebral hemispheres and 1 was in the cerebellum. All 3 patients presented themselves with serious signs and symptoms of an intracranial space-occupying lesion with signs of increased intracranial pressure and of focal lesions of the brain. All 3 cysts were treated surgically. One was simply aspirated on several occasions. One of the others was opened into the lateral ventricle, while the posterior wall of the cerebellar cyst was removed so that it communicated with the cisterna magna. All 3 patients recovered promptly and have been well for many years.

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SURGICAL TREATMENT OF ARTERIOSCLEROTIC OCCLUSION OF COMMON CAROTID ARTERY

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In 1905 Chiari[2] first described a clinical syndrome characterized by temporary hemiparesis, aphasia, or transient loss of consciousness caused by spontaneous occlusion of the internal carotid arteries. Ten years later Hunt[7] again called attention to the same disease and symptom complex and emphasized the importance of careful examination of the cervical carotid vessels in all patients presenting symptoms of possible vascular origin. Not until 1937, however, when Moniz[11] introduced an efficient method of carotid arteriography was accurate diagnosis of the condition made possible, and since that time the disease is being recognized with increasing frequency. The condition is not rare, as evidenced by Hultquist's[6] study of 3,500 autopsies, in which he examined the carotid vessels and evidence of occlusion was found in 91 instances. The majority occurred in the sixth and seventh decades and the condition was about twice as common in men as in women.[16] Recently, Johnson and Walker[8] were able to assemble 107 reported clinical cases. In the great majority the occlusive process occurs in the region of the carotid sinus but in some cases occurs more distally, even as high as the intracranial portion of the internal carotid artery. Interestingly enough, the lesion is 6.5 times more common in the left carotid than the right.[18] In a few cases the lesion is embolic in origin, but the predominant cause is atheroma, which may develop prematurely. The carotid sinus thus appears to be similar to the aortic bifurcation and left coronary artery as a site of predilection for arteriosclerotic occlusion.

Symptomatology may vary widely in severity and mode of onset. Webster *et al.*[19] described four methods of clinical onset for the disorder: (1) explosive, simulating stroke; (2) slowly progressive, characterized by remissions and recurrences (which may suggest multiple sclerosis); (3) with visual symptoms including transient blindness; (4) without signs or symptoms. In addition to the neurologic manifestations already mentioned, Shapiro and Peyton[15] described convulsions and head noises. The latter were described as a soft roaring noise, sensations of escaping steam in the ear, whistling sound or the sound of a watch ticking. Presumably the wide variation of incapacity produced by carotid artery occlusion is related to the acuteness of the occlusive process, presence of collateral vascular supply to the brain, and the completeness of the occlusion.

Several methods of surgical treatment have been advocated and tried

in a few cases, usually with unsatisfactory results. Arterectomy has been performed in the hope that removal of the involved segment might reduce reflex spasm in the cerebral vessels,[1] and cervical sympathectomy[8,15] or denervation of the carotid sinus was advocated for a similar purpose, but in both instances results were disappointing. Definitive treatment by restoration of carotid arterial continuity has been attempted in relatively few cases. Strully[17] attempted thromboendarterectomy for thrombosis of the internal carotid artery in the neck, but was unable to establish a retrograde flow from cerebral vessels and finally ligated the carotid. Although excision of the thrombosed segment with insertion of an arterial homograft would obviously be an effective method, interestingly enough, successful results have not been reported.[4] Indeed, only one satisfactory result from restoration of arterial continuity has been recorded by Eastcott *et al.*[5], who used direct anastomosis of the vessel to the common carotid artery after excision of the partially occluded carotid bifurcation and ligation of the external carotid. After operation the patient was neurologically normal and relieved of intermittent attacks of hemiplegia.

This report is concerned with a case of incomplete occlusion of the common carotid artery at the bifurcation producing annoying head noise and with the method of surgical treatment by means of endarterectomy.

CASE REPORT

D.W., a 71-year-old white man, was admitted to Methodist Hospital on March 4, 1956, complaining of a swishing noise in his left ear. The noise first appeared 4 months previously and had steadily increased in intensity. It was synchronous with heart beat and was most annoying during periods of accelerated heart rate. The noise was accentuated by slight flexion of the head on the neck, particularly if his head were on a pillow, thus interfering with sleep. He denied episodes of numbness, tingling, or unilateral loss of vision, but described dizziness on sitting or standing up. The dizziness was transient, usually lasting less than 1 minute.

Examination. Blood pressure was 150/80 mm. Hg. Normal pulsations were palpable in the common carotid arteries, and a soft thrill was present over the left carotid bifurcation. The left superficial temporal pulse was slightly weaker than the right. On auscultation a loud bruit was audible under the angle of the mandible and was transmitted upward over the superficial temporal artery. Compression of the carotid obliterated the thrill and bruit. Temporary occlusion of the left common carotid artery for 30 seconds (Matas test) produced dizziness, weakness, numbness and tingling sensations in the entire right half of the body, and partial aphasia. All peripheral arterial pulsations were normal.

Laboratory studies revealed a normal hemogram and urinalysis. Electroencephalograms were within the range of normal, and the pattern showed no appreciable change during temporary left carotid occlusion.

Cerebral arteriography was done using Thorotrast solution and showed a partial occlusion of the left common carotid artery beginning just proximal to the bifurcation, with satisfactory filling of the internal and external divisions (Fig. 1). Localized aneurysmal dilatation of the internal carotid artery was demonstrated distal to the stenosis.

Operation. On March 8, 1956, under intravenous Pentothal and endotracheal inhalation anesthesia, cooling of the cranium and brain was attempted by immersing the head in crushed ice. After about 30 minutes this was discontinued, primarily because there was no available method of estimating the degree of local tissue cooling. An oblique incision was made in the neck at the level of the arterial obstruction and the carotid bifurcation was exposed. The occlusive process in the common carotid was located and found to be calcified and fixed, involving only the terminal 2

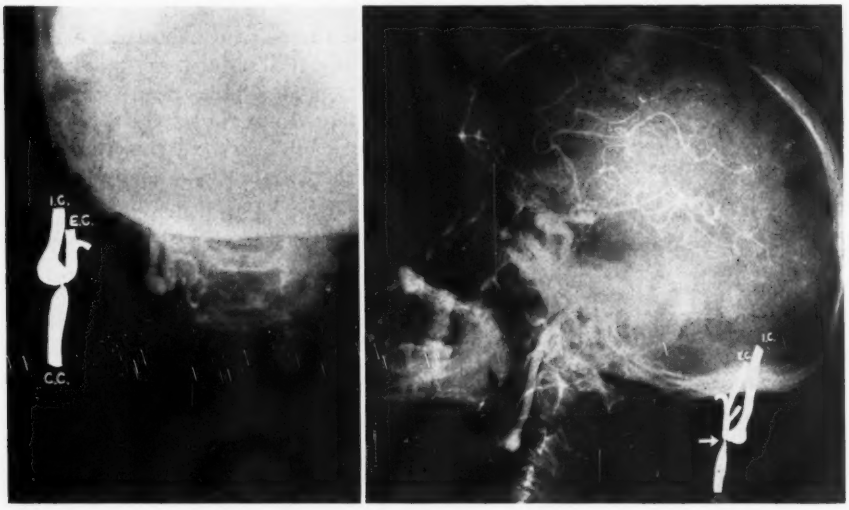

FIG. 1. Carotid arteriograms in anteroposterior and lateral projections showing incomplete occlusion of the common carotid artery at its bifurcation with a post-stenotic dilatation of the proximal portion of the internal carotid artery.

cm. of the common carotid. A strong systolic thrill was palpated in the external and internal carotids, where the arterial tension was palpably lower than in the common carotid. The aneurysmal dilatation of the internal carotid was demonstrated.

A polyvinyl shunt, with needle points at both ends, was used to by-pass the carotid circulation during the period of occlusion (Fig. 2). With the external carotid temporarily occluded, internal carotid flow was maintained by means of the shunt while the atheromatous plaque was removed from the vessel (Fig. 3). The intervening segment of artery was occluded by arterial clamps and a transverse incision was made in the carotid bulb. The occluding calcified arteriosclerotic plaque was peeled out of the lumen by thromboendarterectomy and the arteriotomy was sutured transversely (Fig. 4). Arterial flow was restored after 9 minutes of carotid clamping during which period the shunt continued to function satisfactorily. After arterial repair was complete, strong pulses without a thrill were palpable in the external and internal carotids. The incision was closed without drainage.

Postoperative Course. Soon after awakening from anesthesia the patient announced that the head noises were gone. During the first 12 hours after operation, mild weakness of the right side with thickened speech was present, but these symptoms rapidly resolved, and his recovery was uneventful. He was discharged 6 days

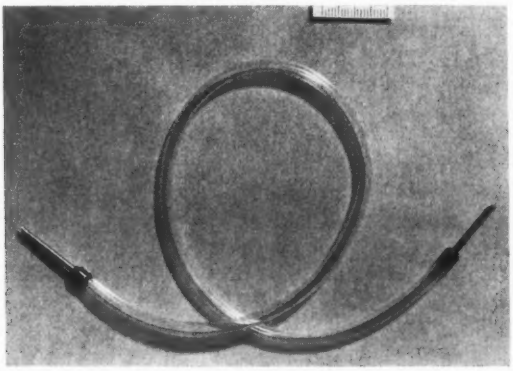

FIG. 2. Photograph of polyvinyl shunt used during period of temporary carotid by-pass showing 14-gauge needle on left and 16-gauge on right.

later with slight impairment of motion and sensation in the right hand. Four weeks later examination revealed normal pulsation in the left common and external carotid arteries without evidence of a thrill or bruit. The slight residual weakness in the right hand was improving and function was almost normal.

DISCUSSION

Although our patient demonstrated no neurologic deficit prior to operation, the results of the carotid occlusion test indicated that cerebral circulation was precariously reduced. Under such circumstances even slight reduc-

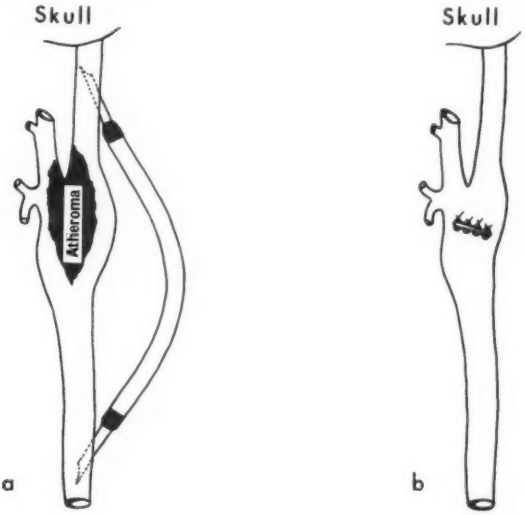

FIG. 3. Drawing showing in (a) method employed with by-pass shunt around the occluded segment and in (b) location of the arterotomy for thromboendarterectomy.

FIG. 4. Photograph made at operation showing the carotid bifurcation after thromboendarterectomy and arterial repair. The hypoglossal nerve is demonstrated crossing the external carotid artery.

tion in cardiac output or periods of hypotension from any cause may produce temporary hemiplegia from ischemia even in the absence of complete carotid occlusion. Furthermore, the narrow lumen of the carotid artery could easily have been occluded acutely by development of a very small thrombus. If operation had been postponed until permanent damage to cerebral tissue had occurred, little could have been expected from restoration of normal carotid circulation. Thus, operation was undertaken in a stage of the disease when carotid occlusion was incomplete. In cases in which symptoms remain transient even though carotid occlusion is complete, operation may also be successful.

Operations upon the carotid artery require temporary occlusion of the vessel during the vascular reconstruction and certain technical measures to protect the brain during the period of temporary ischemia must be employed. The Matas carotid occlusion test provides good indication of the tolerance to temporary ischemia and should always be done before operation in evaluation of such patients. In those patients with total thrombosis of the carotid artery in whom symptoms are transient, the test should be negative, and if operation is done, temporary clamping of the carotid produces no additional cerebral ischemia. On the other hand, when the occlusive process is incomplete, as was the situation in our patient, measures to protect the brain from ischemic damage may be vital to success of the operation. The solution of this technical problem may be achieved either by use of temporary shunts

or by hypothermia. The external shunt used in our patient probably permitted a successful result after 9 minutes of carotid occlusion since the Matas test had demonstrated that neurologic changes on an ischemic basis otherwise appeared after 30 seconds of carotid compression. Arterial flow through one normal carotid artery under basal conditions is estimated to be approximately 250 cc. per minute, but in our patient this rate was probably much less. Measurements on the maximum rate of flow of saline through the shunt at 80 mm. Hg was determined to be 125 cc. per minute. Although this rate of flow is only 50 per cent of estimated basal flow through one carotid artery, reduced flow undoubtedly can protect the brain from serious damage under these conditions. The brief occurrence of right hemiplegia during the first few hours after operation may have been caused by mild ischemic damage, and indicates that the margin of safety with this method may not be sufficient. Perhaps enlarging the lumen of the shunt would have been desirable in this case. The focal residual neurologic change in the right hand also may have been caused by a small cerebral embolus produced in the shunt, although the shunt had been soaked in heparin solution and revealed no evidence of thrombosis. Nevertheless, this complication may indicate the need for continued systemic heparinization after such operations.

Hypothermia may provide another means of solving the problem of cerebral ischemia during temporary carotid occlusion. Hypothermia reduces the metabolic rate of the cerebral tissue and thus reduces the requirements for oxygen and arterial circulation. On the basis of experimental observations hypothermia appears to exert a definite protective influence against ischemic damage.[9,10,12,13] Similar protective influence has been demonstrated in use of hypothermia during temporary occlusion of vascular supply to the spinal cord.[3,14] It is further significant that in the only previously reported successful case of restoration of carotid circulation the procedure was done under hypothermia without any neurologic sequelae. Unfortunately, Eastcott *et al.*,[5] in reporting that case made no mention of a Matas test before operation and it is possible that the patient would have recovered even without hypothermia. Whether local hypothermia of the brain by external cooling is feasible or not is questionable, but it is doubtful that the local cooling was effective in our patient. Local hypothermia of the brain by cooling of the carotid blood has been used clinically and provides a method of producing selective hypothermia to the brain without cooling the entire patient.[9] General body hypothermia was not used in our patient but is undoubtedly a very useful adjunct, particularly in younger patients when the general physical condition permits. The combination of external shunts plus hypothermia offers the maximum protection of the brain during operation on the carotid artery and should be considered the method of choice.

SUMMARY

Arteriosclerotic occlusion of the common or internal carotid arteries is not uncommon and may produce neurologic symptoms of varying types and severity. Onset may be explosive in character, producing hemiplegia, or may

be insidious, producing slowly progressive or transient symptoms. Prompt recognition of the disease and localization of the occlusive lesion permits early treatment and may prevent permanent neurologic symptoms. Surgical treatment with restoration of effective carotid circulation should be employed in all patients if irreversible cerebral damage has not already resulted.

A case is reported of incomplete left common carotid artery occlusion in a 71-year-old man whose only symptoms were annoying swishing sounds in the head. At operation thromboendarterectomy was performed, reconstructing the artery while a temporary external shunt was used to provide cerebral circulation.

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CASE REPORTS AND TECHNICAL NOTES

AGENESIS OF THE SACROCOCCYGEAL REGION

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Congenital anomalies of the sacrococcygeal region associated with neurological abnormalities are not uncommon and sacral agenesis has been reported over 50 times since Hohl's[6] case in 1852. The first clinical description was made by Litzmann[8] in 1885, and Coleschi[2] reported the radiographic appearance in 1918. Díaz Lira[3] described a case in which surgical exploration was done. The present report deals with a case of this abnormality with surgical exploration and electrical stimulation of the roots of the cauda equina for study of the function of the urinary bladder.

CASE REPORT

NCBH #204210. A 6-year-old boy was admitted on May 12, 1955, for investigation of sacrococcygeal abnormality (Fig. 1). He was the third sibling and one of twins. It is not known whether or not the twins were identical. In the third trimester of the pregnancy the mother was found to have lymphosarcoma involving the left hip; however, the pregnancy terminated normally. At birth the patient weighed 4 pounds and there were no initial complications. The child was considered normal except for deformities of the lower extremities. Bilateral equinovarus deformities were corrected surgically and he learned to walk at $2\frac{1}{4}$ years. He did not achieve control of bladder or bowel. At the age of 4 a transurethral resection of the neck of the bladder was done with some improvement of bladder capacity but with no improvement of the sphincter. He was considered to be mentally normal at the age of 6 years.

Family History. The twin died at 4 weeks of age of an acute infection of the urinary tract. No roentgenograms were made and bilateral hydronephrosis and hydroureters were found at postmortem examination. No special studies of the spine were made.

Physical Examination. Temperature was 98.6°F., blood pressure 94/60, pulse rate 86, and respiratory rate 20. The abnormal findings were limited to the lower extremities, genitalia, and sacral region. The anterior muscles of the thigh were well developed and strong, while the buttocks were flat and atrophic. Below the knees the legs tapered down to the ankles with no visible muscles. The arches of the feet were high with the toes fixed in a flexion deformity. Scars of the previous orthopedic procedures were evident on the feet. The anal sphincter was relaxed. He dribbled urine intermittently.

Neurologic Examination. He was an alert, bright boy of 6 years. The cranial nerves were intact. There was good flexion and adduction at the hips but poor extension. Voluntary flexion and extension of the knee were normal but there was only a flicker of movement in the ankles and toes. The deep reflexes were brisk and equal in the upper extremities, but the knee jerks were barely present and the ankle jerks were absent. Cutaneous plantar stimulation on each side caused a quick voluntary withdrawal of the leg with slight flexion of the toe. All

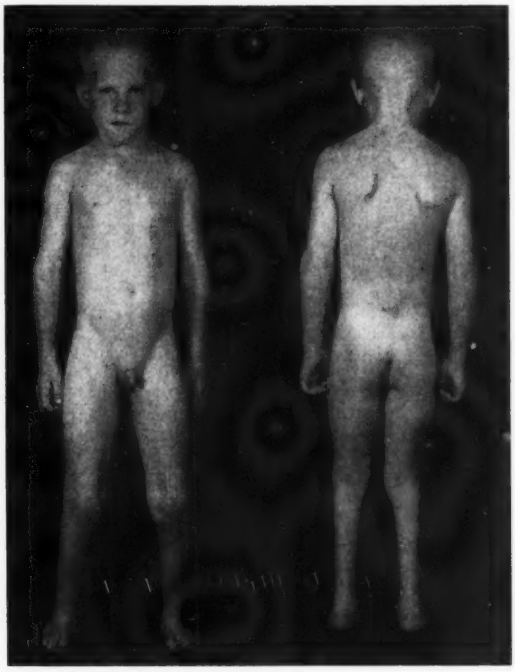

FIG. 1. Photograph of sacrococcygeal abnormality in a 6-year-old boy. Note tapering lower extremities and flattened gluteal regions.

sensation was normal including the cutaneous areas in the region of the motor deficit. There was no subjective sensation of bladder on filling the bladder. He occasionally experienced rectal sensation with defecation.

Accessory Clinical Findings. Blood counts and urine were normal; serological test of the blood for syphilis was negative. Roentgenograms revealed bilateral cervical ribs with fusion abnormalities of the C5, C6, C7, and possibly the T1 vertebrae (Fig. 2). There were five lumbar vertebrae and the sacrum was absent with the iliac bones meeting at the midline (Fig. 3). The hip joints were normal. Roentgenogram of the chest showed the lungs to be clear; the size of the heart was reported to be at the upper limit of normal, and there was slight prominence of the shadow of the pulmonary artery. Excretory urograms showed normal renal pelves and calyces. A cystogram revealed a small contracted bladder with reflux of dye into the right ureter. Cystometrograms done in the waking state were unsatisfactory. A closed system could not be maintained because of leakage around the catheter and a small contracted bladder. A lumbar puncture was attempted in the lower lumbar region but was not successful.

Operation. On July 11, 1955, a complete laminectomy was performed of L2 through L4. The dural cul-de-sac was found to terminate at the L2-L3 interspace. The nerve roots below this level were embedded in a loose fatty connective tissue and the manner of exit of the roots from the canal was normal (Fig. 4). Faradic stimulation (1–2 volts) of the nerve roots produced active motor responses in the hamstring muscles bilaterally. A cystometer was connected with the bladder through a urethral catheter but there were no contractions on electrical stimulation of the sacral nerve roots. The dural sac was opened and cerebrospinal

FIG. 2. Roentgenogram of cervical spine showing bilateral cervical ribs, with fusion abnormalities of C5, C6, C7, and possibly T1 vertebrae.

fluid flowed freely. The conus medullaris was not seen but a rubber catheter could be passed cephalad 10 cm. without meeting resistance in the subarachnoid space. The dura mater and wound were closed.

Postoperative Course. The wound healed *per primam* and there was no alteration in the neurologic status.

Three months after operation the parents reported that in the morning he could void 1 or 2 ounces of urine with a good stream. He had required no enemas, although occasionally he was incontinent of feces and continued to wear special waterproof pants.

DISCUSSION

The striking configuration of the sacral region and the lower extremities, which taper down to the feet with equinovarus deformities, identifies this abnormality. This appearance has been described as "mermaid-like," or "sirenen" by Feller and Sternberg.[4] The buttocks and sacrococcygeal region are flat with prominent iliac crests, shortened intergluteal fold, and iliac bones that touch one another in the midline. Freedman[5] reviewed 45 cases and reported 8 cases (18 per cent) in which the hips were dislocated; club feet were present in over half of the cases.

Bony abnormalities of the spine can occur from the cervical to the sacral region. These may consist of hemivertebra, abnormal fusions, failure of development of the neural arches, sacralized and wedged 5th lumbar vertebra and congenital aplasia. Congenital synostosis of several ribs, genu recurvatum, and congenital subluxation of the knees also have been described. The relation of the dural sac to the vertebral

FIG. 3. Anteroposterior and lateral roentgenograms of lumbar spine and pelvis showing absence of sacrum and coccyx.

canal may be altered, as in our case, with the cul-de-sac ending at the L2–L3 level. This would explain the failure of the attempted lumbar puncture at the lower level. Lausecker[7] reported a sacral deformity in a 24-year-old man who had bowel and urinary incontinence. The patient died of acute infection of the urinary tract. There was striking deformity of the L4 and L5 vertebrae with a rudimentary sacrum. The lower lumbar canal was narrowed and deformed, but he stated that the spinal cord ended at the L1 level and the nerves issued normally from the canal. From observation of the photograph and drawing in his case it is conceivable that the bony deformities compressed the cauda equina (Figs. 5 and 6).

A multiplicity of visceral abnormalities may be associated with this condition. In one review of 45 cases,[5] there were 16 (35 per cent) instances of associated geni-

tourinary or bowel involvement, usually rectal, and there was 1 case of transposition of the viscera. White[11] described the postmortem findings in a case of sacral agenesis with an accessory spleen, an umbilical hernia filled with bowel, undescended right testicle, and median kidney with two suprarenals. The right ureter was normal; the left was a fine impervious cord. In our case there were no visceral abnormalities evident, but the dead twin was known to have had bilateral hydronephrosis and hydroureters. It is not known whether or not the twin had agenesis of the sacrum and coccyx.

Fecal and urinary incontinence of varying degrees are the rule. In our case cystometrogram done at the time of surgery under general anesthesia revealed a small contracted bladder and electrical stimulation of the nerve roots produced no contraction. The reason for the apparent improvement in bladder function after laminectomy is not known, although Díaz Lira[3] has made a similar observation.

Motor defects predominate over sensory with associated atrophy of the muscles below the knees and in the sacral region. Walking may be possible in some patients if the associated anomalies of the bones and joints are not severe. In our case the child walked as if on stilts,

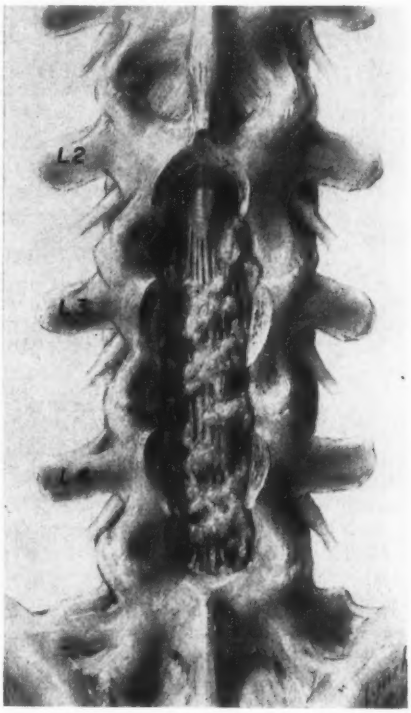

FIG. 4. Drawing of lumbar area exposed by laminectomy.

using his thigh muscles; however, he had a remarkable degree of agility in spite of his deformities.

Sensory findings have been listed as "confused," "chaotic," or "incomplete." The sensory defect when it exists is much less than one might anticipate in view of the profound motor deficit; careful testing of our patient revealed normal sensation in a cooperative child. The discrepancy between the obvious large motor deficit and the normal sensory function remains unexplained.

Reflexes were usually absent below the knees and mass reflexes from cutaneous stimulation have not been reported.

Surgical exploration of this type of abnormality was first reported by Díaz Lira[3] in a 9-year-old boy with sacrococcygeal agenesis. He expressed the opinion that the cauda equina was compressed. The spinal canal was exposed from L3 to S1. The spinous process of the 4th lumbar vertebra was formed by two bony masses separated by a tight fibrous band and beneath this the nerve roots were entangled in a fibro-

fatty mass. The nerve roots could not be separated from the mass. Postoperatively the child retained some urine and had the desire to urinate, voiding approximately 20 cc. At the end of the third postoperative day a cathartic was given and the patient expressed the desire to defecate. From that time he had no fecal incontinence,

FIG. 5. Photograph from Lausecker's[7] case, showing multiple deformities of the lumbar spine. (Reproduced by courtesy of Dr. H. Lausecker.)

regained urinary control and retained 40 cc. for several hours. A later examination revealed normal anal reflexes with good function of the sphincters. Páez[10] reported a case of subtotal sacrococcygeal agenesis and mentioned two others in which there was improvement after transurethral resection of the neck of the bladder. He also referred to a case of Heras in which the superior hypogastric plexus was resected and another case in which the cauda equina was decompressed.

In view of the experiences cited above, surgical exploration is indicated to obtain further information regarding function of the bladder in the individual patient. Some improvement has been reported and there may be variation in innervation of the bladder from patient to patient as in those with lumbosacral myelomeningocele. The possibility of improving function of the bladder in this type of abnormality

by altering the motor supply of the bladder awaits further trial in selected cases.[1]

A wide range of variation exists in the congenital abnormalities of the sacrococcygeal region, from a minimal defect such as spina bifida occulta to the more severe

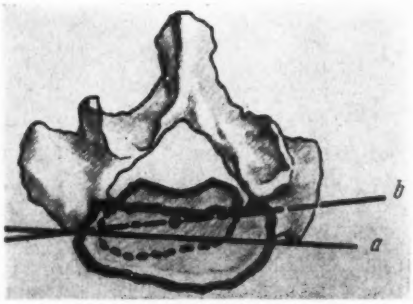

FIG. 6. Illustration from Lausecker's[7] case, showing abnormality of the vertebral bodies and narrowing of vertebral canal. (Reproduced by courtesy of Dr. H. Lausecker.)

deformities associated with sacrococcygeal agenesis. We believe that these deformities are more prevalent than indicated in the literature and know of 5 unpublished cases reported to us by Martinat.[9]

SUMMARY

A case of sacrococcygeal abnormality is reported with surgical exploration and electrical stimulation of the lumbosacral nerve roots. This abnormality consists of minor bony defects of the cervical and lumbar spine with absent sacrum and coccyx. There was a severe loss of motor function below the knees but normal sensation. Rectal and bladder incontinence improved subjectively after laminectomy.

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LARGE CALCIFIED CRANIOPHARYNGIOMA AND BILATERAL SUBDURAL HEMATOMATA PRESENT AT BIRTH

SURVEY OF NEONATAL BRAIN TUMORS

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This report is made to record the occurrence in a newborn not only of a large calcified intracranial mass (craniopharyngioma) but its concomitant association with bilateral subdural hematomata developing *in utero*. A survey of the literature has failed to reveal another patient in whom a craniopharyngioma was present at birth. The earliest case found was one mentioned in a report by Jackson.[9] In his paper of 1916 he referred to a case reported by Lawson in 1887. The patient was a 3-month-old male infant. Two references to craniopharyngioma occurring in children of the age of 2 years have also been found.[6,8] The rarity of a large calcified tumor at birth and the presence of bilateral subdural hematomata are self-evident. A survey of other brain tumors occurring in the newborn will be made.

CASE REPORT

A neurosurgical consultation was requested at St. Joseph Mercy Hospital in Detroit, Michigan by the pediatrician in attendance who recognized an enlarged head and bulging widely patent fontanelles in a newborn full-term male delivered on April 24, 1955. He showed no other physical defects. The pregnancy had been normal, and the child had two normal siblings. The delivery was uncomplicated, but low forceps was employed. Immediately following birth the condition of the child was considered poor, and artificial respiration was instituted subsequent to which he was placed in an incubator. The birth weight was 7 pounds and 13 ounces.

Examination. When first seen by the writer the child was 2 days of age and showed no neurological deficit except for the enlarged head (circumference 43 cm.). He cried somewhat feebly, but was otherwise active and alert. There was marked palpable separation of suture lines and bulging of intracranial contents between all ununited bone plates.

Course. The initial clinical impression was of marked hydrocephalus developing *in utero*. However, bilateral fontanelle taps at the age of 7 days revealed that the condition apparently was caused by bilateral subdural hematomata. Sixty-three cc. of yellow fluid were easily aspirated from the right subdural space, and 58 cc. of yellowish somewhat sanguineous fluid were similarly removed from the left subdural space, resulting in marked scaphoid depression of the anterior fontanelle. The size and color of these hematomata at such a short time following birth, plus the existence of an enlarged head at birth seemed to indicate that these clots had existed *in utero* and had not occurred as a result of parturition. Attempts to discover a blood dyscrasia as an explanation of the development of hematomata *in utero* were unrewarding and became unnecessary when roentgenograms of the skull revealed the large calcified intracranial mass shown in Fig. 1. The presence of this mass and its movement with *in utero* movements of the fetus could well explain a tear of bridging subdural veins and consequent development of subdural hematomata.

During the next 4 weeks bilateral fontanelle taps with drainage by needle of the hema-

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FIG. 1. Large suprasellar calcified tumor in infant at age of 9 days.

tomata were done on six different occasions. After the fourth tapping the left hematoma was dry and remained so. However, the right-sided one continued to accumulate. Beginning at the fourth week of life further tapping of the right subdural hematoma failed to reduce the bulging anterior fontanelle to a scaphoid state, as had occurred previously, and the growth of the head increased more rapidly (from 43 to 46 cm. in the 2-week period) suggesting abrupt development of obstructive hydrocephalus. The mass itself was needled through the fontanelle on two occasions. The first time this was done was at 11 days of age, but only 1–2 cc. of bloody fluid could be obtained and no significant tissue was removed. At $2\frac{1}{2}$ weeks of age the mass was again needled and 5 cc. of straw-colored oily fluid were obtained. The infant was discharged home on May 26, 1955.

2nd Admission. The baby was admitted to Mt. Carmel Mercy Hospital in Detroit, Michigan on June 1, 1955. He now weighed 9 pounds, but the head had grown to 49 cm. in circumference. Otherwise the baby seemed more vigorous and alert than it had previously.

The left fontanelle tap on June 1, 1955 was dry, but 45 cc. of straw-colored fluid were removed from the right subdural space without depressing the fontanelle.

On June 3, 1955, 43 cc. were removed from the right subdural space through the anterior fontanelle, and at the same time 8 cc. of black-colored fluid was aspirated from within the calcified mass and replaced with an equivalent amount of air. Roentgenograms at this time showed the air confined within the calcified mass in a scattered fashion without outlining any single loculated cyst.

On June 7, 1955 a right subdural tap allowed removal of 20 cc. of straw-colored fluid. At the same time the right ventricle was tapped, and 5 cc. of clear colorless fluid were removed and replaced with air. Roentgenograms showed a dilated right ventricle displaced far to the right and not communicating with the left lateral or third ventricles (Fig. 2). It was of inter-

est incidentally that the mass had now shifted to the right of the midline whereas it was located primarily to the left of the midline in the first films taken when the infant was 10 days old. The tumor also appeared to have enlarged by about 25 per cent of its original size.

The likelihood of accomplishing anything worth while surgically in this infant was obviously remote. Yet there being nothing else to offer the patient or parents, the decision to undertake surgical removal of the tumor regardless of the consequences was made. Pre-

FIG. 2. Postero-anterior view of air in right lateral ventricle, which is displaced far to the right by calcific mass.

operative preparation with cortisone was thought unnecessary by the pediatrician now in attendance. The infant was now 7 weeks of age, weighed approximately 9 pounds, and the circumference of the head was 49 cm.

Operation. On June 11, 1955, under endotracheal anesthesia, through a small right frontal scalp flap an osteoperiosteal bone flap was turned down. A thin subdural hematoma and membranes were present beneath the dura mater and these were gently wiped away. The brain bulged moderately, but the convolutions were not flattened. A 1-inch cortical incision was made in the superior right frontal gyrus and at a depth of 1 inch the displaced right ventricle was entered. In apposition to its medial wall the hard surface of an encapsulated tumor was encountered. A frozen section biopsy revealed it to be a craniopharyngioma (Fig. 3). It was not cystic, but it shelled easily from surrounding brain tissue and was relatively avascular. Staying within the tumor it was relatively easy to remove large fragments of it without more than minimal hemorrhage. The infant tolerated the procedure well until removal of the last fragment of tumor. This fragment was the posterior shell of the tumor lying in apposition to the third ventricle and hypothalamic structures, all of which had been displaced far posteriorly to a position overlying the tentorium. The optic nerves and internal carotid arteries were markedly elongated, running posteriorly from their point of origin in the region of the sella turcica to an extent of about 1½ inches. The tumor had pressed into the sella turcica, but even so the optic nerves appeared to be more elongated than flattened out by pressure.

During removal of the tumor the left lateral ventricle was also entered and, as with the right ventricle, it was easily separated from the surgical field with cottonoid strips. The tumor had grown up between the lateral ventricles and separated them. At no place was the tumor adherent to surrounding brain or basal structures. When the final fragment of the posterior shell of the tumor was gently teased away from the third ventricle and hypothalamus, the infant immediately stopped breathing and could not be revived. There was no large hemor-

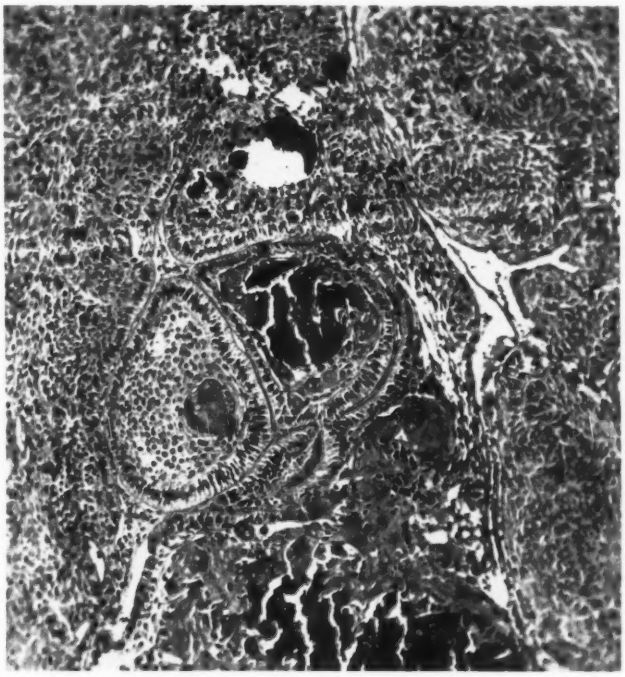

FIG. 3. Low-power magnification of tumor showing calcific areas and stratified squamous epithelium.

rhage at this or at any other time during the operation. The infant received about 300 cc. of blood during the 3-hour procedure as replacement for its estimated loss of blood.

DISCUSSION

The interest in this case centers around the occurrence at birth of an intracranial tumor. The presence of calcification in a tumor in a newborn, the incidence of this histological type at birth, and the concomitancy with bilateral subdural hematomata are by themselves unique features. Neonatal intracranial neoplasms are so uncommon, however, that the peculiarities of this case should not overshadow completely a consideration of this entire group. Arnstein *et al.*[1] reviewed this subject in 1951 while reporting an intraventricular cavernous hemangioma in a 3-day-old infant. In a survey of the literature they were able to find 13 other cases of brain tumors occurring during the neonatal period (birth to 60 days). An additional search has disclosed 16 other cases of tumors manifesting themselves clinically before the age of

60 days, not included in their study or reported since that publication appeared.[2,4,5,7,10,11,13,14] Together with this case, therefore, a total of 31 cases of neonatal intracranial neoplasms have now been found on record. In 10 of the 17 added cases the tumors were teratomas and most of these were reported in obstetrical literature as a cause of dystocia. Four of the 13 tumors in the review by Arnstein *et al.*[1] were also teratomas. Thus, 14 of the 31 neonatal brain tumors reported were teratomas. Of this group, it is known that 9 produced dystocia and delivery was accomplished only after opening the head *in utero*. Five of the infants were born alive, 1 surviving, however, for only one-half hour. In the remaining 16 cases, exclusive of the 1 herein reported, 3 tumors were classified as probable medulloblastoma, 2 as sarcoma, and 1 each as dermoid, cystic blastoma, pineoblastoma, ependymoma, atypical polar spongioblastoma, glioblastoma multiforme, neuroepithelioma, choroid plexus papilloma, meningioma, cavernous hemangioma, and one simply as glioma.

It is known that 13 of the entire group of 31 patients were dead at birth or immediately thereafter. There were only 2, other than the 1 reported here, who underwent intracranial surgery. In 1 of these an enormous calcified parieto-occipital meningioma was only biopsied, and the patient expired 6 days later. In the other, the patient expired following the second choroid plexectomy for what was thought to be a communicating hydrocephalus. At autopsy a pineoblastoma was found. Only in the present case was an effort made for cure by total removal of the tumor. The specimens in all other patients were obtained at autopsy.

Although certain of the living patients presented focal neurological signs and symptoms, such manifestations were exceptional. The most common feature was an enlarging head which in several instances occurred with great rapidity. Many of the tumors, particularly the teratomas, were so large that intra-uterine perforation of the head was necessary to enable delivery. In these cases and in certain of the others the tumors had grown to such size as to nearly entirely replace brain. Tumors that were smaller showed no predilection for location. They were found within, imbedding, and between hemispheres, within the mid-brain or the cerebellum, and several were intraventricular.

Besides the present case, there were 2 others in which the tumor showed calcification by roentgen ray. In 1 of these, a case of Cushing's,[3] the patient was a 2-month-old infant who appeared normal at birth but a rapidly enlarging head developed. Roentgenograms revealed irregular calcified areas scattered throughout the cranial chamber. Aside from needling the lesion and draining off cystic fluid, no surgical procedure was done. The lesion at autopsy proved to be a huge teratoma. The other infant was one reported by Cuneo and Rand[2] who was operated upon at the age of 3 months after a 2-month history of vomiting and opisthotonic spells. Roentgenograms showed a lacy calcification in the left parieto-occipital area with a superimposed, circumscribed thickened skull. The tumor proved to be a meningioma and after biopsy was considered too large for attempted removal. In neither of these cases, apparently, was the tumor and its extent as sharply delineated by the calcium as in the case reported here. The calcification and its apparent growth in this patient raise a provocative speculation concerning the fetal age when this tumor began. If during the 7 weeks of life the tumor enlarged in size by 25 per cent, one might presume by retrograde interpolation that the tumor at birth was at least 6 months old. That would fix the onset of tumor at less than 3 months' fetal age. Rathke's pouch first begins to develop in the fourth week of fetal life.[12] From another point of view it is now evident that it is possible, at least in an infant, for a calcified tumor to grow

to the size of a baseball in less than 9 months and that the long duration usually attributed to most calcified intracranial tumors is not necessarily indicated. This is perhaps more valid for children in whom increased calcium metabolism in conjunction with growth processes is more apt to occur.

It appears, therefore, that of the tumors presenting at birth or shortly thereafter about half prove to be teratomas and often cause dystocia sufficient enough to force perforation of the head for delivery. Judging by this case and the experience of others the prognosis even with surgical intervention seems to be hopeless. Many of these cases, however, appeared before the availability of modern neurosurgical and anesthetic techniques.

Since several of the tumor types that occurred were enucleable lesions, with prompt recognition of the etiology surgical cure of certain neonatal brain tumors may yet be possible if they are accessible. Identification of the lesion may be made more easily if, as in some of these cases, calcification is present. A brain tumor, therefore, must be considered in the differential diagnosis in the presence of intracranial calcification in a newborn. Hydrocephalus that develops with great rapidity may be another clue indicating a tumor.

SUMMARY

A case report is made of a large calcified craniopharyngioma present at birth in conjunction with bilateral subdural hematomata. Its relationship to other neonatal brain tumors reported in the literature is discussed.

I wish to acknowledge with gratitude assistance given in the preparation of this report by Mrs. Clara Cziske.

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THE LATERAL SITTING POSITION

FOR OPERATIONS IN THE POSTERIOR FOSSA AND IN THE CERVICAL AND UPPER THORACIC REGIONS OF THE SPINE

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(Received for publication March 15, 1956)

Three positions, the upright or sitting,[1] the lateral,[2] and the prone, are in use nowadays for operations in the cerebellopontine angle and in the cervical and high thoracic regions of the spinal canal. In our experience, the latter is the least convenient and the first offers definite advantages over the other two. However, the upright or sitting position presents serious difficulties when the need arises to lower the patient's head should the blood pressure fall to dangerous levels. This situation has become more pressing with the recent advent of potentialized anesthesia.

It is obvious that in operations in the posterior fossa or cervical column with the patient in the upright or sitting position, lowering the head is an awkward maneuver since it demands stopping the procedure, with great risk of contamination of the

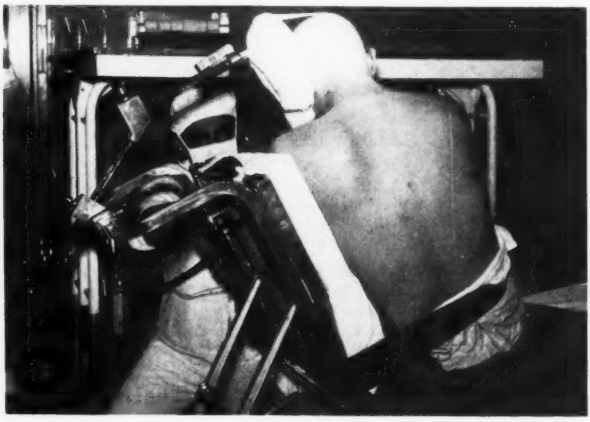

FIG. 1. Anesthetist's approach to the patient. Note the regular headrest.

wound, and necessitates redraping the field to continue the operation. This is avoided by using what we call the "lateral sitting position" (Fig. 2) which, if necessary, permits the neurosurgeon to complete the operation either in the lateral position (Fig. 3) or back in the sitting position if this is considered safe. In other words, the patient can be easily shifted from the sitting to the lateral position or vice versa without having to redrape the field to continue the procedure once the circulatory condition allows it. All degrees of inclination can be reached between the almost

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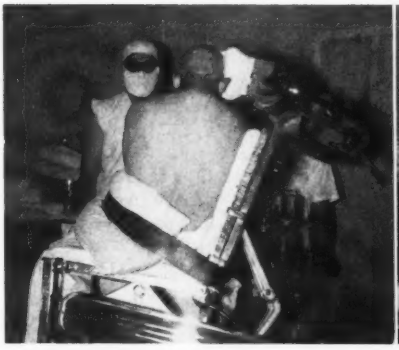

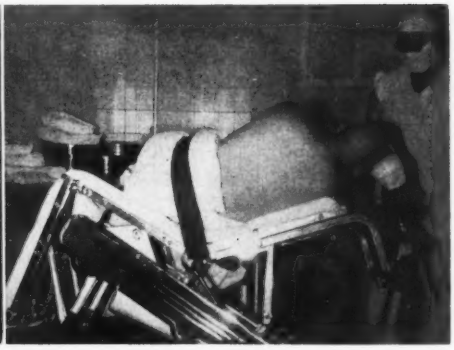

FIG. 2 (*left*). Patient in the lateral sitting position. Fig. 3 (*right*). Patient brought down to the lateral horizontal position.

vertical and the horizontal plane and even below this if it becomes necessary. In addition to these advantages, the lateral sitting position requires, in our experience, less specialized equipment and can be more easily obtained.

DESCRIPTION

The patient sits sideways on the regular operating table previously set up as a chair and well protected by a thick mattress. A pillow is placed underneath the knees and another around the hips. A long, strong leather belt over this pillow ties the patient to the backpiece of the table which is slightly tilted backwards. The feet rest on another pillow over a stool which can be lowered or raised at will. Both arms are kept forward and downward and the supporting shoulder ought to be elevated to the upper edge of the backpiece. Children are raised with a stool and pillows (Figs. 4 and 5). In case of a lateral lesion, the head rests on the cerebellar headpiece on the side opposite to that of the pathology. Flexion of the head and

FIGS. 4 and 5. A 5-year-old child in the lateral sitting position.

neck is favored by the action of gravity and it is regulated by a convenient adjustment of the headpiece which should be properly padded with rubber. The distribution of body weight facilitates the maintenance of a correct position all through the procedure.

The instrument table shown in Fig. 1 is placed in front of the patient at the same level with his head. It allows the anesthetist to have free access to the patient under the drapes and it can be easily lowered and displaced with the head should this have to be brought down. This type of table can be substituted by Mayo stands.

We use our standard operating and instrument tables and, at first, in a number of operations, we employed the regular headrest (Fig. 1). Lately, we have been using a wooden piece attached to the cerebellar headrest with the aid of small straps (Figs. 2, 3, and 4).

COMMENT

We have found this position most convenient for the following procedures: cerebellopontine angle tumors, section of the 5th, 8th, 9th, and sensory portions of the 10th cranial nerves, posterior fossa operations performed through midline incisions (tumors, and certain by-passing procedures), and tumors, herniated discs, chordotomies, and rhizotomies in the cervical and high thoracic (above T7) regions of the spinal canal.

SUMMARY

The lateral sitting position is a modification of the regular upright or sitting position. We believe that it has all the advantages of the latter and, in addition, it is easier to achieve, requires less specialized equipment, and affords the facility of being able to lower with ease the patient's head in case that this should be needed.

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A SIMPLE CASSETTE CHANGER FOR CEREBRAL ANGIOGRAPHY

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Cerebral angiography has gone through several stages of development since its introduction in the late 1920's. At first great difficulty was encountered in the development of a contrast material suitable for injection into the cerebral circulation. Difficulties were also encountered in developing proper methods of injection into the carotid system. The problem of securing films of good radiological quality of a correctly positioned head has at times been almost overwhelming. These difficulties were magnified by the surgical drapes necessary for open arteriography. With the more recent development of techniques of percutaneous arteriography using relatively nontoxic injection materials, the procedure has become of more practical value. However, it was apparent early that a series of films was better than a single film and a large number of very complicated pieces of apparatus have been developed to achieve this end. It seems obvious, however, that the ideal apparatus has not yet been built. Most of the apparatus thus far have been cumbersome, expensive and not always reliable.

In 1952, an attempt was made to design a device to hold standard roentgen-ray cassettes for a lateral exposure of the skull during angiography. We were able to build a simple apparatus of plastic materials. Shortly thereafter, a similar model of wood was constructed in a home workshop, using materials costing less than ten dollars (Fig. 1). This same model has been used in the Department of Radiology at Passavant Memorial Hospital for a total of several hundred arteriograms with no problems of maintenance or mechanical failure. Structurally similar models have also been in use in several other hospitals. With either of these models, it has been possible to take a series of three 10×12 inch lateral films of the skull as rapidly as the film cassettes can be removed, possibly in as little as 5 seconds or at any other time intervals desired. With slight alterations it would be possible to take a similar number of films in the anteroposterior projection.

The apparatus consists of a "L"-shaped frame with films for the lateral view being held in the vertical portion. A series of lead-backed cassettes are used and these are pushed against a Lysholm grid by a spring-backed pressure plate. After the first exposure the cassette is pulled from the apparatus by a tab of adhesive tape previously fixed to its side. The next cassette is then automatically pushed into position. Only the first and second cassettes need be lead-backed. In actual practice, a preliminary film of the skull is taken before any injection is made to check the function of the roentgen-ray apparatus and position of the head as well as to spot any foreign objects, such as hair pins. The successful use of this apparatus requires some team work between the surgeon and the roentgenologist as to the timing of the injection in relation to the roentgen-ray exposures and there must be synchroniza-

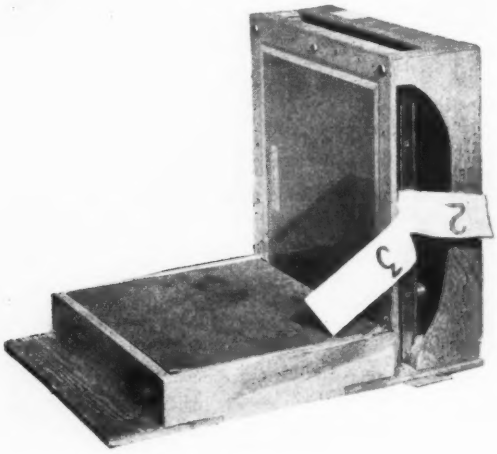

FIG 1

tion of action between the technician who controls the roentgenological unit and whoever may be removing the cassettes. The anteroposterior films may be taken using either the Potter-Bucky apparatus beneath the roentgen-ray table or by the use of grid cassettes beneath the patient's head. The use of this apparatus makes it possible to obtain three good straight lateral films with a minimum amount of effort. The whole series of films are quickly available. There is no necessity for a wait of 30 minutes or more as with some types of apparatus using roll film.

In the construction of this apparatus some experimentation may be necessary to achieve proper spring tension behind the pressure plate and some tailoring-to-fit will be necessary to achieve smooth motion of the cassettes. Exact dimensions are not given since these depend upon the size of the cassettes used. However, once the proper fit is obtained, the apparatus has been found to function well without alterations.

A greater number of films taken in shorter periods of time may well be desirable but it is our belief that this apparatus functions better than do other devices costing many times more. It has served well as a stop-gap until the development of a reliable reasonably simple seriagraph.

NOTICES

AMERICAN COLLEGE OF SURGEONS

The 42nd Clinical Congress of the American College of Surgeons will be held in San Francisco, October 8–12, 1956. Neurosurgical sessions will take place at the Fairmont Hotel. Two Surgical Forum sessions will be devoted to reports of research in the field of neurosurgery, on October 10 at 1:30 P.M. (Dr. Howard C. Naffziger presiding) and on October 11 at 8:30 A.M. (Dr. C. Hunter Shelden presiding). On October 12, at 9:00 A.M., there will be panel discussions on "The surgical management of intracranial aneurysms and arteriovenous malformations" (Dr. Russell Meyers, moderator) and "Surgical management of cerebrovascular disease not including aneurysms and arteriovenous malformations" (Dr. E. S. Gurdjian, moderator), and at 1:30 P.M. clinical neurosurgical papers will be presented.

AMERICAN ELECTROENCEPHALOGRAPHIC SOCIETY

At the 10th annual meeting of the American Electroencephalographic Society held in Atlantic City, June 15–17, 1956, the following were elected to office: President, Dr. John R. Knott, Iowa City, Iowa; President-elect, Dr. Robert S. Dow, Portland, Oregon; Secretary, Dr. J. K. Merlis, Boston, Massachusetts; Treasurer, Dr. Peter Kellaway, Houston, Texas.

AMERICAN NEUROLOGICAL ASSOCIATION

At the 81st annual meeting of the American Neurological Association held at the Claridge Hotel, Atlantic City, June 18–20, 1956, the following officers were elected for the year 1956–57: President, Dr. H. Houston Merritt; President-Elect, Dr. Israel S. Wechsler; 1st Vice-President, Dr. Richard B. Richter; 2nd Vice-President, Dr. Augustus Rose; Secretary-Treasurer, Dr. Charles Rupp; Assistant Secretary, Dr. William F. Caveness.

The next annual meeting of the Association will be held at the Claridge Hotel, Atlantic City, New Jersey, June 17–19, 1957.

ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASE

The annual meeting of the Association for Research in Nervous and Mental Disease will be held on December 7 and 8, 1956 at the Hotel Roosevelt, New York City. The subject of the meeting will be "The Brain and Human Behavior."

CONGRESS OF NEUROLOGICAL SURGEONS

The 6th annual meeting of the Congress of Neurological Surgeons will take place at the Palmer House in Chicago, November 1–3, 1956. The theme of the program is centered on the pituitary body: its ophthalmologic, endocrine and surgical aspects.

Dr. Wilder Penfield will be the principal guest speaker. He will present two addresses. The first will concern the function of the temporo-sylvian fissure, and in the second he will present a historical sketch, Dawn of Medicine in the Aegean.

Inquiry should be directed to the office of the Secretary-Treasurer, Dr. Philip D. Gordy, 1007 Delaware Avenue, Wilmington, Delaware.

JOURNAL OF NEUROSURGERY

The *Journal of Neurosurgery* is devoted to the publication of work relating primarily to neurosurgery, including studies in clinical neurophysiology, organic neurology, ophthalmology, radiology and pathology. Short articles on unusual cases and on technique, special instruments or equipment that might be useful to others in the field are also acceptable.

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Articles: Name of author, full title of article, name of periodical, year, volume, first and last page of article. Abbrevations of names of periodicals should conform to the style used in *A world list of scientific periodicals published in the years 1900–1933* (Oxford University Press, 1934).

Books: Name of author, full title of book, city, publisher, year, edition, full pagination (if book is in more than one volume, give the number of volumes).

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